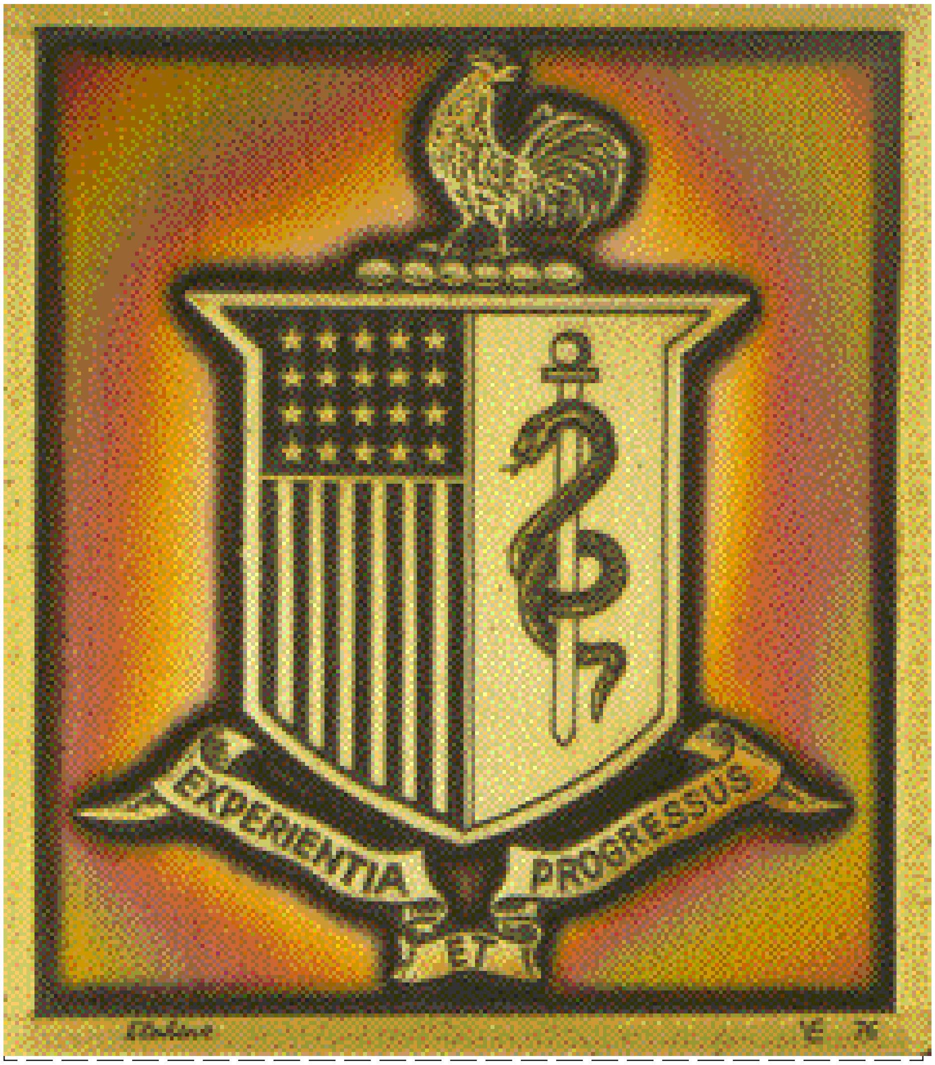
REHABILITATION OF THE INJURED COMBATANT
VOLUME 2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The Coat of Arms
1818
Medical Department of the Army

A 1976 etching by Vassil Ekimov of an
original color print that appeared in
The Military Surgeon, Vol XLI, No 2, 1917

The first line of medical defense in wartime is the combat medic. Although in ancient times medics carried the caduceus into battle to signify the neutral, humanitarian nature of their tasks, they have never been immune to the perils of war. They have made the highest sacrifices to save the lives of others, and their dedication to the wounded soldier is the foundation of military medical care.

Textbook of Military Medicine

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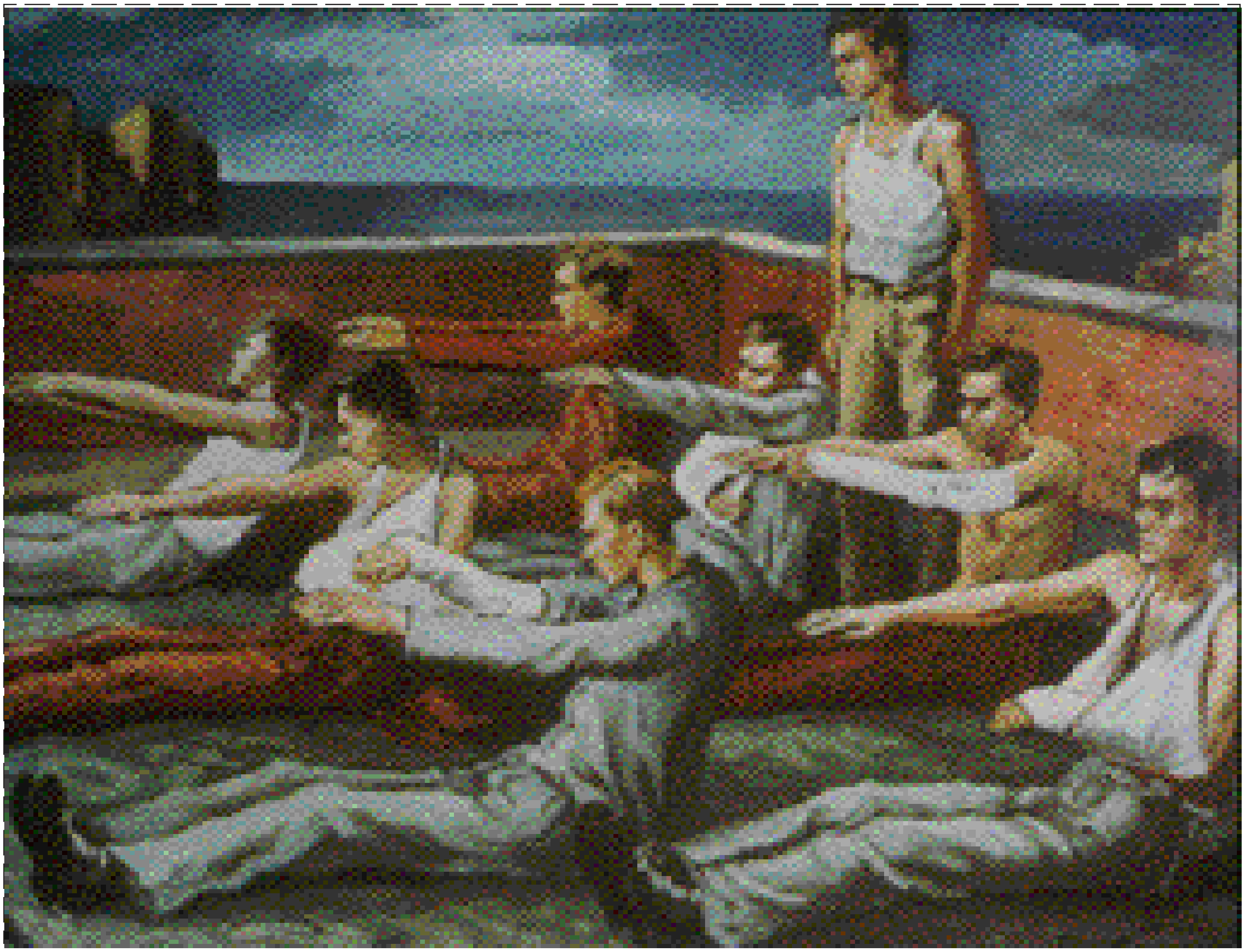

Exercise Period for Wounded

Artist: Marion Greenwood ca. 1945

Art: Courtesy of US Center of Military History, Washington, DC.

REHABILITATION
OF THE
INJURED COMBATANT
VOLUME 2

Specialty Editors

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Contents

Foreword by The Surgeon General xi

Preface xiii

Patient Flow in a Theater of Operations xv

Section II: Rehabilitation of the Injured Combatant 417

9. Peripheral Nerve Injuries 419
10. Rehabilitation Management of Burn Casualties 575
11. Orthotics for the Wounded Combatant 703
12. Prevention of Immobility Complications Through Early Rehabilitation 741

Section III: Special Contributions 779

13. Physical Fitness and Physical Training for Military Performance 781
14. Physiatry: Interdisciplinary Management 829
15. Vocational Rehabilitation and Community Reintegration of the Wounded Combatant 845
16. The US Army Physical Disability System 863

Acronyms and Abbreviations xvii

Index xxi

Foreword

Highly skilled soldiers, sailors, airmen, and marines in today's military services possess knowledge of weapons systems that requires considerable training to acquire. These combatants are difficult to replace should they become severely injured during conflict or training. Providing the best medical care possible to America's military sons and daughters and returning these highly trained individuals to duty are, therefore, dual needs of paramount importance. Rehabilitation plays an essential role in the return to duty through both exercise, which prevents contractures, and beginning ambulation at the earliest possible time after injury, which prevents the deleterious effects of bedrest.

Lieutenant Colonel Howard A. Rusk, MD, the US Army Medical Corps officer in World War II who introduced active rehabilitation into Army Air Corps hospitals, and one of the founders of modern rehabilatative medicine, observed that "men did not get ready for full duty playing blackjack or listening to the radio."[1(p463)] It was apparent to physicians in World War I and World War II that physical rehabilitation was supremely important. In order to return to duty, aggressive therapies were necessary soon after injury. For those unable to return to duty immediately, early rehabilitative intervention prevented the effects of immobility and maximized the patients' functional potential.

Rehabilitation must be thought of as a continuum of care spanning the time from shortly after injury to full functional restoration. It is a common misconception that rehabilitative care should be relegated largely to Veterans Affairs hospitals. This argument has been made in the past, but we need only look at historical experience to realize that rehabilitation must begin soon after injury—while the patient is still being treated in military hospitals. During World War II, for example, the army established amputation centers where the highest quality rehabilitation could be provided. But in 1946 the Special Exhibit Committee for Rehabilitation stated:

> Delay in inaugurating rehabilitation procedures is the most frequent cause of failure. If there is too much delay in instituting a program of rehabilitation, muscular atrophy, fixation of joints, and mental depression may progress to a point at which complete restoration becomes impossible.[2(p497)]

Far too often the hard-earned lessons of war are forgotten between conflicts. Physical medicine and rehabilitation developed as a specialty as a direct consequence of the great conflagrations of the two world wars. Although medical science has progressed at a phenomenal pace, more than 50 years have elapsed since the last book of rehabilitation specifically regarding war injuries, *Rehabilitation of the War Injured,* was published in 1943. It is fitting that the vastly improved diagnostic and therapeutic rehabilitation interventions be consolidated in the *Textbook of Military Medicine,* a series that will constitute an encyclopedia of combat casualty care. For this reason, this textbook, *Rehabilitation of the Injured Combatant,* will be a valuable reference for the physicians and allied providers who care for those who are injured while fighting for our nation.

Lieutenant General Ronald R. Blanck
The Surgeon General
U.S. Army

April 1998
Washington, DC

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1. Rusk HA. The growth and development of rehabilitation medicine. *Arch Phys Med Rehabil*. 1969;Aug:463–466. Editorial.
2. Special Exhibit Committee on Physical Medicine of the American Medical Association. Exhibit on physical medicine: Physical therapy, occupational therapy and rehabilitation. *Arch Phys Med*. 1946;Aug:491–498.

Preface

> This nation has no more solemn obligation than healing the hurts of our wounded and restoring our disabled men to civil life and opportunity. The Government recognizes this and the fulfillment of the obligation is going forward fully and generously. ... It is merely the payment of a draft of honor which the United States of America accepted when it selected these men, and took them in their health and strength to fight the battles of the Nation. They have fought the good fight; they have kept the faith, and they have won. Now we keep faith with them, and every citizen is endorser on the general obligation.
>
> —Woodrow Wilson[1(pv)]

These words from the past ring as true today as they did in 1919. It is the responsibility not only of our nation but also of the medical corps of all the services to ensure the very best care possible for all combatants. This best possible care includes the responsibility to provide the highest-quality rehabilitative care when a soldier, sailor, airman, or marine has sustained a potentially disabling condition. For this reason, a textbook on rehabilitation is considered essential by the Borden Institute.

Rehabilitation traces its roots to the two world wars. The tremendous needs of injured combatants with amputations, severe hand injuries, spinal cord injuries, brain injuries, burns, and nerve injuries stimulated development of this field, which includes physiatry (physicians specializing in rehabilitation), physical therapy, and occupational therapy. As modern warfare has drastically improved its lethality, medicine has also improved its ability to save lives. But during their recuperative phase, almost all those with war wounds need at least strengthening to prevent complications of immobility, and range of motion exercises to prevent contractures.

Medical literature is replete with textbooks on the rehabilitative care of civilians. This textbook, however, focuses on the aspects of care that are specifically related to wounds sustained through combat and military training, for almost all of these require some component of rehabilitation to ensure full functional restoration. The textbook is published in two parts and organized into three sections. The first section introduces the field of rehabilitation, its history, and its functions in the modern military. The second section, the largest and most comprehensive, deals with injury-specific rehabilitation: of burn wounds, nerve injuries, spinal injuries, the special problems of amputees, and so forth. The authors of these chapters produced comprehensive treatises far beyond any preconceived expectations. They have captured the essence of modern rehabilitation and its application to the military. The chapter on preventing complications of immobility is an important contribution; all military physicians and healthcare providers must understand these important principles. The third section deals with exercise and training in ways to prevent injuries, yet maximize performance and strength. In addition, the army's medical boarding system has been outlined admirably and will guide the reader through this complicated system.

In the modern military services, which make substantial investments in training their personnel, vocational restoration encompasses returning to active duty. Depending on the needs of the military, the current national situation, and the special skills possessed by the injured combatants, rehabilitation to the point of return to duty can be an important source of force reconstitution during a conflict.

(Preface continues)

The efforts of the two specialty editors of this textbook, Timothy R. Dillingham, MD, and Praxedes V. Belandres, MD, Colonel, Medical Corps, U.S. Army, have made this two-part volume a reality, and I thank them for their determination to provide nothing less than the best for those in their care. *Rehabilitation of the Injured Combatant* is a welcome addition to the *Textbook of Military Medicine* series. This volume symbolizes the armed forces' commitment to providing the finest rehabilitative care possible to those who "fought the good fight" in service to their nation and in so doing sustained grievous injuries.

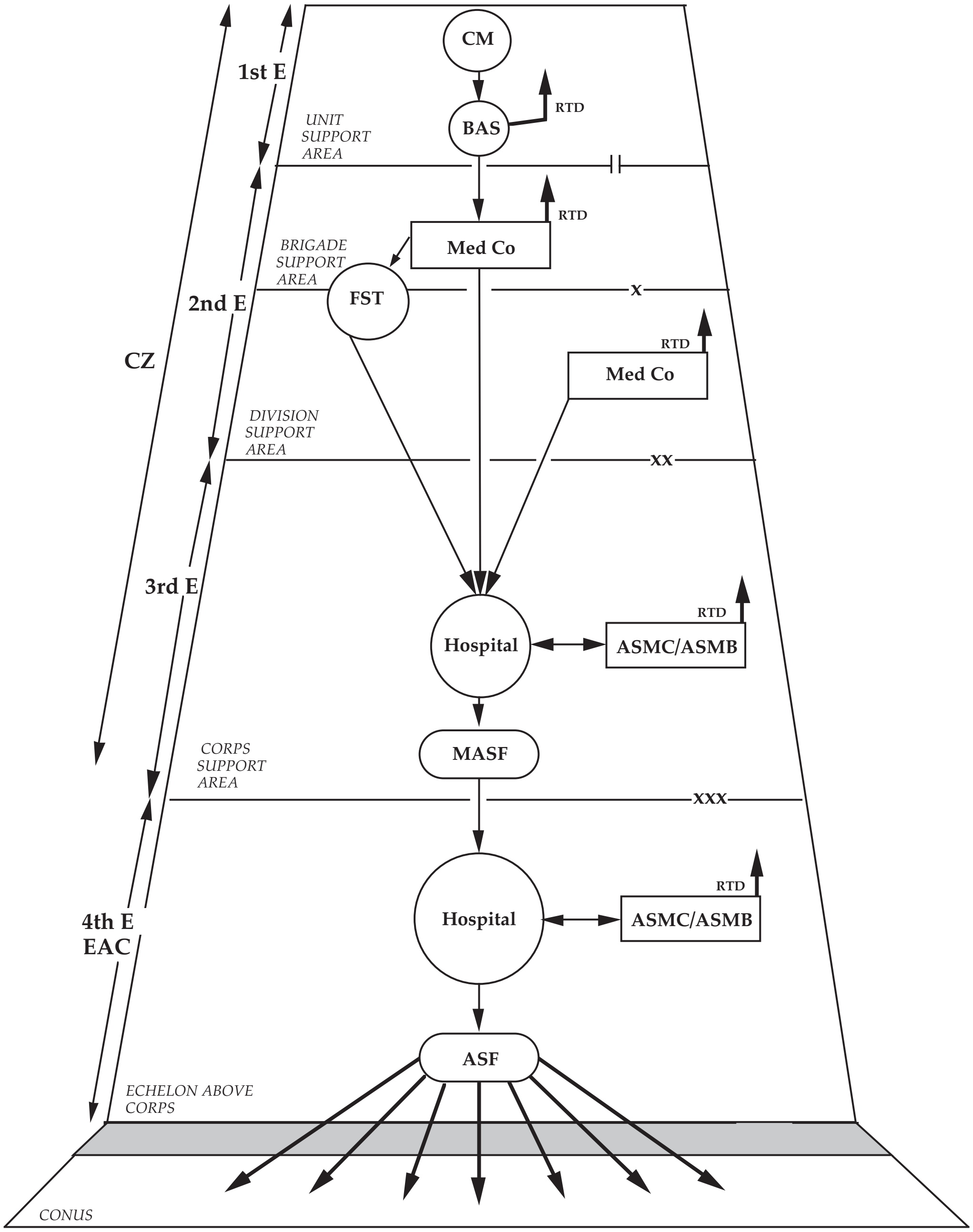
Brigadier General Russ Zajtchuk
Medical Corps, U.S. Army

April 1998
Washington, DC

1. Wilson W. Epigraph. In: Harris G. *The Redemption of the Disabled: A Study of Programmes of Rehabilitation for the Disabled of War and of Industry*. New York, NY: D. Appleton and Co; 1919: v.

The current medical system to support the U.S. Army at war is a continuum from the forward line of troops through the continental United States; it serves as a primary source of trained replacements during the early stages of a major conflict. The system is designed to optimize the return to duty of the maximum number of trained combat soldiers at the lowest possible echelon. Far-forward stabilization helps to maintain the physiology of injured soldiers who are unlikely to return to duty and allows for their rapid evacuation from the battlefield without needless sacrifice of life or function.

Medical Force 2000 (MF2K)
PATIENT FLOW IN A THEATER OF OPERATIONS


CM
BAS
RTD
1st E
UNIT SUPPORT AREA
Med Co
RTD
BRIGADE SUPPORT AREA
FST
x
2nd E
CZ
Med Co
RTD
DIVISION SUPPORT AREA
xx
3rd E
Hospital
ASMC/ASMB
RTD
MASF
CORPS SUPPORT AREA
xxx
Hospital
ASMC/ASMB
RTD
4th E
EAC
ASF
ECHELON ABOVE CORPS
CONUS

ASF:	Aeromedical Staging Facility, USAF	E:	Echelon
ASMB:	Area Support Medical Battalion	EAC:	Echelon Above Corps
ASMC:	Area Support Medical Company	FST:	Forward Surgical Team
BAS:	Battalion Aid Station	MASF:	Mobile Aeromedical Staging Facility, USAF
CM:	Combat Medic	Med Co:	Medical Company
CONUS:	Continental United States	RTD:	Return to Duty
CZ:	Combat Zone		

Chapter 9

PERIPHERAL NERVE INJURIES

MICHAEL D. ROBINSON, M.D.*; AND PHILLIP R. BRYANT, D.O.†

INTRODUCTION

EPIDEMIOLOGY

PERIPHERAL NERVE ANATOMY AND PHYSIOLOGY

PATHOPHYSIOLOGY OF NERVE INJURIES

END ORGAN CHANGES FOLLOWING DENERVATION

MECHANISMS OF NERVE INJURY

ELECTRODIAGNOSIS OF PERIPHERAL NERVE INJURIES

REHABILITATIVE MANAGEMENT OF PERIPHERAL NERVE INJURIES

CAUSALGIA (COMPLEX REGIONAL PAIN SYNDROME, TYPE-II)

UPPER EXTREMITY NERVE INJURIES AND ENTRAPMENT SYNDROMES

NERVE INJURIES AND ENTRAPMENT NEUROPATHIES IN THE LOWER EXTREMITY

NERVE INJURIES OF THE FOOT AND ANKLE

CONCLUSION

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INTRODUCTION

Nerve injuries are likely to have occurred since the most ancient of recorded battles. Blunt and lacerating trauma to nerves was almost certainly inflicted in alarming numbers in early hand-to-hand combat. It is probable that upper extremity peripheral nerves, including the brachial plexus, were common sites of injury in these ancient conflicts. Mechanized warfare of the current century has also resulted in nerve injuries ranging from mild, isolated nerve trauma to severe injury at multiple sites. Many nerve injuries occur directly from blunt trauma or lacerations, while others are secondary complications due to fractures or compartment syndrome.

Combat-related nerve trauma was well documented in World Wars I and II, as well as in Korea and Vietnam. The experience of neurosurgeons in World War I was recorded by Pollock and Davis.[1] Their text, which contains information on peripheral nerve anatomy, as well as technical methods of nerve exposure, mobilization, transplantation, and suturing, was a popular reference for general surgeons and neurosurgeons involved in World War II.[2]

Woodhall et al[2] note that the most important lesson learned from the World War II experience in managing peripheral nerve injures was application of early nerve repair. Delay was abandoned in favor of prompt exploration within 21 to 90 days after nerve insult in all nerve injuries where any doubt existed regarding spontaneous regeneration. The World War II experience taught neurosurgeons that concomitant soft tissue, bone, and vascular injuries critically affected the timing and techniques of nerve repair.

Electrodiagnosis was in its infancy during World War II. Although it was recognized that it had potential in helping to identify the distribution and severity of nerve lesions and in detecting changes associated with regeneration, electrodiagnosis was only marginally available to physicians during World War II. In addition, at that time, the machines and techniques for assessing nerve integrity were unsophisticated and limited in their applications. A substantial number of soldiers in the Persian Gulf War who were treated by military physical medicine and rehabilitation services were also noted to have sustained peripheral nerve injuries.[3] Many of these soldiers suffered profound and sometimes multiple nerve damage. Although some recorded nerve injuries steadily improved with time, some were so severe that reinnervation activity and functional return were markedly compromised or nonexistent.

Proper management of peripheral nerve injuries requires an understanding of the distribution and function of nerves, the mechanisms of nerve injury, and an appreciation of the regeneration process. It is imperative to astutely examine the patient to obtain the clinical clues necessary for accurate diagnosis. Detection and localization of nerve injury can be complicated in the presence of multiple additional trauma such as fractures or vascular compromise. Carefully planned and performed electrodiagnostic studies based on the history and examination offer additional diagnostic and prognostic information.

Among the most important aspects of care is proper rehabilitation of nerve injuries, regardless of whether surgical intervention is required. Rehabilitation of nerve injuries invariably requires an interdisciplinary approach in order to obtain optimal nerve recovery and function. A physiatrist, neurosurgeon, neurologist, orthopedic surgeon, or plastic surgeon, as well as physical therapist, occupational therapist, and vocational specialist, are among the team members typically involved in the rehabilitative effort.

EPIDEMIOLOGY

Registries of peripheral nerve injuries compiled during the World Wars have shed important light on the severity of injuries, distribution of nerves injured, and associated injuries. Out of nearly 175,000 casualties sustained by American Expeditionary Forces in World War I, 3,129 injuries—nearly 2% of total battle injuries—were documented nerve injuries. Statistical analyses were performed on 2,390 of these, focusing on the nerves most commonly injured. Injuries severe enough to warrant surgical exploration accounted for approximately 45%. A majority, 55%, were felt to be of lesser severity and operative intervention was not undertaken. Of the 1,088 operative cases, 266 were found to be in at least partial continuity. All told, 66% of nerve injuries were partial, that is, they were noted to be anatomically or functionally in continuity. Thirty-four percent were complete injuries (Table 9-1) requiring surgical coaptation.

Injuries of the sciatic nerve accounted for 23%; radial nerve, 21.5%; peroneal nerve, 17%; median nerve, 11%; tibial nerve, 1.5%; ulnar neuropathies, 21%; and nonspecified, 5% (Table 9-2).[4]

The incidence of peripheral nerve injuries among those injured in combat during World War II has been estimated to be somewhat higher than during the first World War. Spurling and Woodhall[5] suggested 5% to 8% [assuming 500,000 World War II approximately 30,000] to be more likely. Similar to World War I, the percentage of nerve injuries severe enough to warrant surgical exploration was just under 50%, a rate consistent in both the Mediterranean and European theaters. A representative cross section of cases that required surgical exploration when treated in the Mediterranean theater revealed that 40% had complete injuries, 24% had partial injuries, and 36% were in continuity. When combined with those cases not severe enough to require surgical exploration, nearly 80% manifested functional or anatomic continuity. Surgical reconstruction was warranted in only 20%.[5(pp237,238)]

In the European theater, 6,245 peripheral nerve injuries were observed between D-day and V-E day. Again, 2,873 or 46% were severe enough to warrant surgical exploration. Of all nerve injuries, 1,528 or 25% required surgical reconstruction. Nerve injuries in anatomic or functional continuity comprised 75%[5(p248)] (see Table 9-1).

TABLE 9-1

SEVERITY OF NERVE INJURIES

Severity	World War I (%)	World War II* (%)	World War II†(%)
Complete (Explored)	34	20	25
Partial (Not Explored)	55	50	54
Partial (Explored)	11	30	21
Total partial nerve injuries	66	80	75
Percentage of all injuries	2	5-8	NR

NR: not recorded
*Mediterranean Theater, World War II
†European Theater, World War II

TABLE 9-2

PERCENTAGE DISTRIBUTION OF PERIPHERAL NERVE INJURIES

Nerve	World War I	World War II*	Persian Gulf†
Ulnar	21	28	12
Radial	21.5	20	11
Median	11	15	18
Musculocutaneous	NR	0.75	NR
Axillary	NR	0.75	NR
Brachial Plexus	NR	8.5	10
Sciatic	23	8	7
Peroneal	17	11	16
Tibial	1.5	3	8
Femoral	NR	2.5	2
Saphenous	NR	1	NR
L/S Plexus	NR	NR	10
Cranial	NR	NR	3
Not Specified	5	1.8	3

NR: not recorded
*Mediterranean Theater, World War II
†Physical Medicine Service, Persian Gulf War

In the Mediterranean theater, injuries to the ulnar nerve were most common, accounting for 28%; radial nerve, 20%; median nerve, 15%; peroneal nerve, 11%; sciatic nerve, 8%; brachial plexopathies, 8.5%; tibial nerve, 3%; femoral nerve, 2.5%; saphenous nerve, 1%; musculocutaneous nerve and axillary nerves each, 0.75%; and nonspecified, 1.8% (see Table 9-2). Multiple nerve injuries were common, with an incidence ranging from 8% to 20%.[5] The upper extremity manifested multiple nerve injuries more commonly than the lower extremity.

Data from the Mediterranean theater revealed compound fractures to be associated with peripheral nerve injuries in over one third of the cases. Humeral fractures were associated with radial, median, or ulnar nerve injuries 40% of the time. A similar percentage of forearm fractures was related to nerve injuries. Assessment of all nerve injuries requiring surgical correction also showed a 33% rate of association with fractures (Table 9-3).

Vascular injuries not severe enough to cause gangrene were associated with 21% of all nerve inju-

TABLE 9-3

RATES OF CONCOMMITANT INJURIES WITH PERIPHERAL NERVE INJURIES IN WORLD WAR II*

Injury	Percentage
Compound fractures	33
Humeral fractures	40
Radial/Ulnar fractures	39
Vascular injuries	21

*Mediterranean Theater, World War II

ries sustained in the Mediterranean theater (see Table 9-3). A lower rate of 8% was documented when casualties undergoing surgical intervention from all theaters and the zone of the interior were examined.

In the 1990 Persian Gulf War, a substantial number of soldiers, who were treated by Physical Medicine and Rehabilitation services, sustained peripheral nerve injuries. Nerve injuries were associated with 68% of penetrating wounds, 67% of amputations, and 58% of fractures. The high percentages likely reflect the referral pattern of the population seen by the Physical Medicine and Rehabilitation service. Electrodiagnostic evaluation was the primary reason for referral in 40%.

In the Persian Gulf War, median nerve injuries were most common, comprising 18% of injuries. Peroneal nerve injuries followed at 16%; ulnar nerve injuries at 12%; radial nerve injuries at 11%; brachial plexopathies at 10%; lumbar plexopathies at 10%; tibial nerve injuries at 8%; sciatic neuropathies at 7%; femoral nerve injuries at 2%; and cranial neuropathies at 3%[3] (see Table 9-2).

While statistics conveying the magnitude of combat-related nerve injuries are essential, one must also consider that even larger numbers of nerve injuries and neuropathies treated during the world wars were not sustained during battle. When radiculopathies are included with all other peripheral nerve injuries and nerve-associated pain disorders, over 34,000 admissions were logged during World War II that were not battle related.[6] The cumulative loss of manpower and productivity because of such disorders likely impacted on effectiveness and readiness of the fighting force. Thus, a sound understanding of both traumatic and atraumatic nerve injuries is essential for comprehensive management to be undertaken.

PERIPHERAL NERVE ANATOMY AND PHYSIOLOGY

Neuroanatomy

The neuron is the fundamental anatomic unit of the peripheral nervous system (Figure 9-1). Each neuron consists of a cell body (soma), dendrites, and an axon. The cell body contains the nucleus, numerous mitochondria, and essential apparatus for the production of structural proteins. Neurotubules and microfilaments are transported distally and provide the edificial construct that imparts rigidity to the relatively fluid axon. Neurotransmitters are also produced in the cell body and are transported along the axon to synaptic boutons.

Dendrites are branching appendages emanating from the cell body. The multiple phalanges and small spines provide an immense surface area with which to interface with and receive stimuli from other neurons. The chemical signal imparted to the dendrites is transformed into an electrical signal. The membrane potential courses through the cell body, where all the numerous inputs summate, allowing for control of neuronal discharge along the axon.

The axon is an extension of the cell body that arises from the axon hillock. The hillock is devoid of myelin and is likely the summation point for neuronal impulses.[7] Axon length varies from a few millimeters to several feet. Classically, nerve fibers have been divided into unmyelinated and myelinated groups based on the characteristics of axonal ensheathment. The inextricable intercellular relationship between axons and Schwann cells becomes apparent at this level. Developmentally, Schwann cells originate from neural crest cells.[8] The cells develop with the extending neurons. Characteristically, 8 to 15 unmyelinated fibers are circumferentially surrounded by a single Schwann cell, each 200 to 500 μm in length.[7] A column of Schwann cells encases the entire length of the fiber group. The boundaries of individual Schwann cells are difficult to discern due to the myriad interdigitating cytoplasmic phalanges binding the adjacent cells together. Fifty percent to 80% of all nerve fibers are unmyelinated.

Anatomically, myelinated fibers differ from unmyelinated fibers in several ways. One axon is surrounded by one layer of Schwann cells. The cytoplasm of each Schwann cell becomes attenuated and wraps repeatedly around the axonal segment. Ulti-

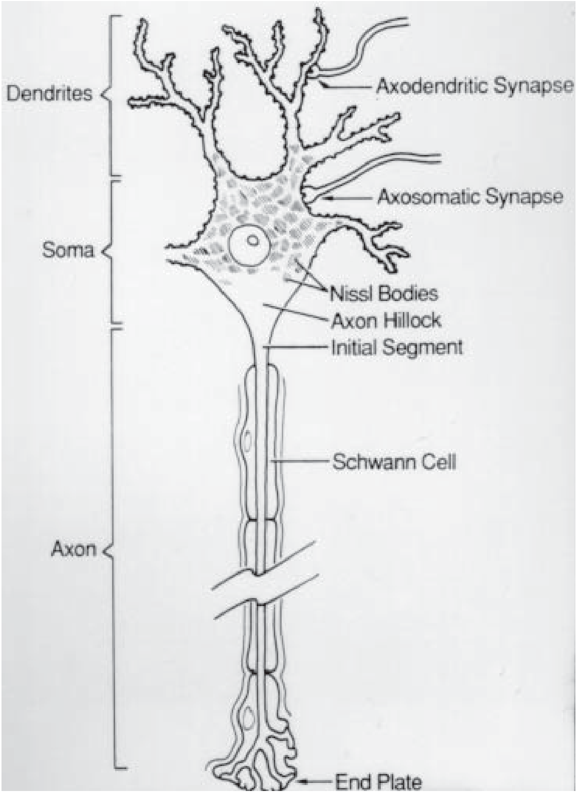


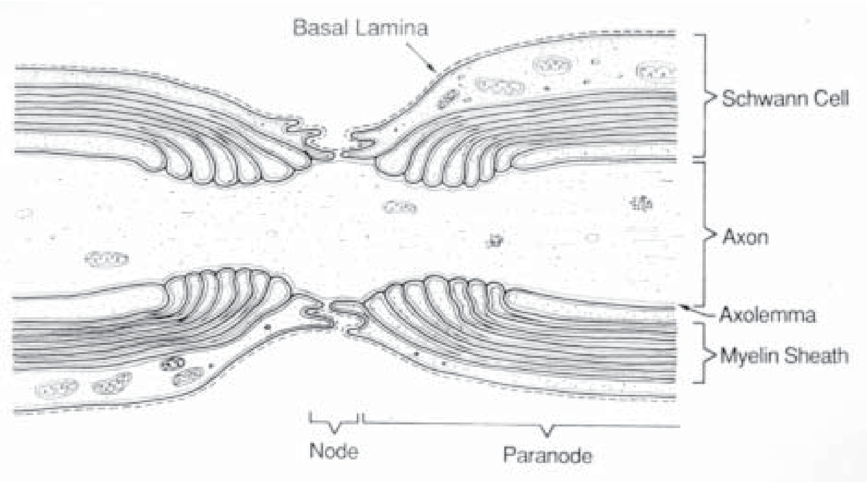
Fig. 9-1. The neuron, including dendrites, soma, and myelinated axon. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure, Function and Reconstruction.* New York: Raven Press**;** 1990**.**

mately, a multilayered, laminated, lipoprotein sheath is formed. The myelin layer divides the neuron into segments ranging from 0.1 mm to 1.8 mm in length,[9] each forming an internode. These internodes are high resistance, low capacitance insulators that greatly enhance the speed with which an impulse propagates down the length of an axon. The unmyelinated, intersegmental focuses, termed nodes of Ranvier, are highly specialized apparatuses uniquely suited to effect saltatory conduction (Figure 9-2). Sigworth[10] noted that at the nodes, large concentrations of voltage-sensitive sodium ion channels are present. The Schwann cell collar surrounding the edges of the node are rich with mitochondria and may provide the energy mechanism necessary for function of the ionic pumps.[11]

Impulse Propagation

Rapid fluctuations in ionic concentrations traveling along the axolemma predicate neuronal signaling. Embedded in the cell membrane are ion channels, selectively permeable to potassium, sodium, and chloride as well as other ions such as calcium (Ca^{++}) (Figure 9-3). Movement of ions through these channels allows for the establishment of an electrical potential across the cell membrane. A predominance of potassium (K^+) channels allows a relatively large concentration of K^+ to diffuse along its concentration gradient out of the cell. The diffusion of K^+ is held in check by the electrical forces generated by impermeable anions trapped inside the cell, which eventually attract the K^+ back into the cell. An equilibrium between the opposing forces is established. The cell membrane is also permeable to Na^+. The concentration gradient for Na^+ is opposite that noted for K^+. High extracellular concentrations of Na^+ follow the gradient and are attracted into the cell. The anionic forces within the cell also draw Na^+ ions inward. A deluge of Na^+ rushing into the cell is prevented by a relatively lower permeability of the cell to Na^+ ions. How-

Fig. 9-2. The node of Ranvier. Myelin sheaths form collars at either end of the node, replete with mitochondria, which facilitate impulse transmission. The basement membrane limits ionic diffusion in areas adjacent to the node. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure. Function and Reconstruction.* New York: Raven Press; 1990.



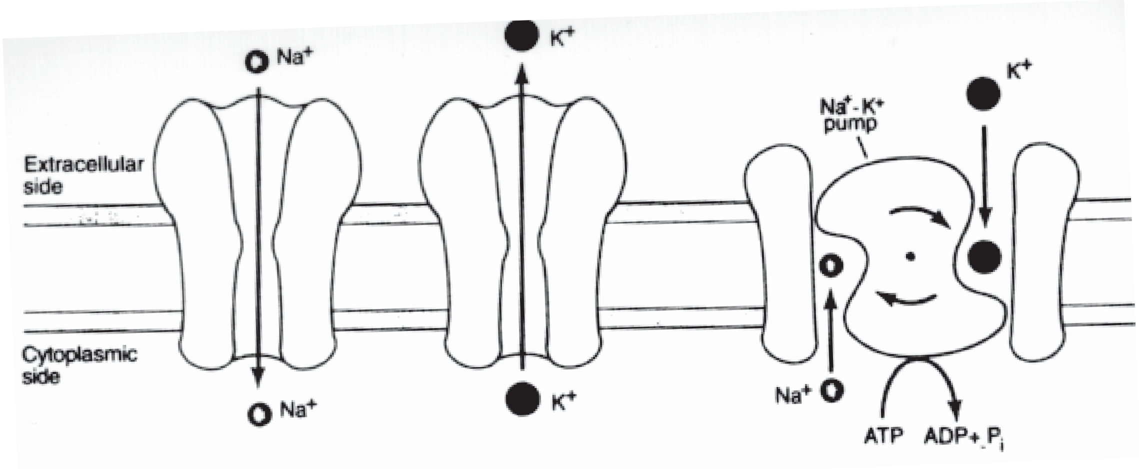


Fig. 9-3. The passive fluxes of Na^+ and K^+ into and out of the cell are balanced by an ATP-dependent Na^+–K^+ pump, which drives Na^+ out of the cell and K^+ into the cell. Adapted with permission from Koester J. Membrane Potential. In: Kandel ER, Schwartz JH, Jessel TM, eds. *Principles of Neural Science.* 3rd ed. Norwalk, Conn: Appleton and Lange; 1991: 87.

ever, as Na^+ flows in, the intracellular fluid becomes less negative and the more freely permeable K^+ is driven out of the cell. A steady state is reached with the electrical gradient across the membrane holding near –60 mV. While the general flux of Na^+ and K^+ are balanced ionically, concentrations must be held in check, otherwise all the intracellular K^+ would be depleted, and the ever-rising intracellular Na^+ would eventually reduce the membrane potential and permanently depolarize the cell. Maintaining the membrane potential requires energy as Na^+ and K^+ are respectively pumped out of and into the cell against their concentration gradients. The Na^+-K^+ ATPase (adenosine triphosphatase) dependent pump extrudes three Na^+ ions from the cell for every two K^+ it brings into the cell.[12] The larger number of ions purged is incompletely offset by the ease with which K^+ diffuses back into the cell. An equilibrium is reached at a resting membrane potential near –70 mV.

Any factor changing the careful balance of ionic flow into or out of the cell will lead to a rapid change in membrane potential. In addition to the nongated ionic channels previously mentioned, the cell membrane also possesses gated ionic channels. While it has not been completely elucidated, that gating mechanism is thought to be positively charged molecules coupled to the lipid matrix that repel cations. Any change in the electrical potential across the membrane will alter the relative strength of the gate (ie, as the inside of the cell becomes less negative, the gate becomes relatively less positive, and its repellent force is diminished).

Tremendous increases in Na^+ permeability occur as the membrane potential becomes less negative (Figure 9-4).The gated ionic Na^+ channels become more permeable when the cell is depolarized by approximately 7mV (ie, to –63mV).[12] Na^+ rushes into the cell down ionic and concentration gradients. As the membrane potential approaches –55mV, the firing level is attained and complete depolarization of the cell occurs. The Na^+ channels quickly close following this rapid depolarization, which is limited to +45mV. Gated K^+ channels then open,

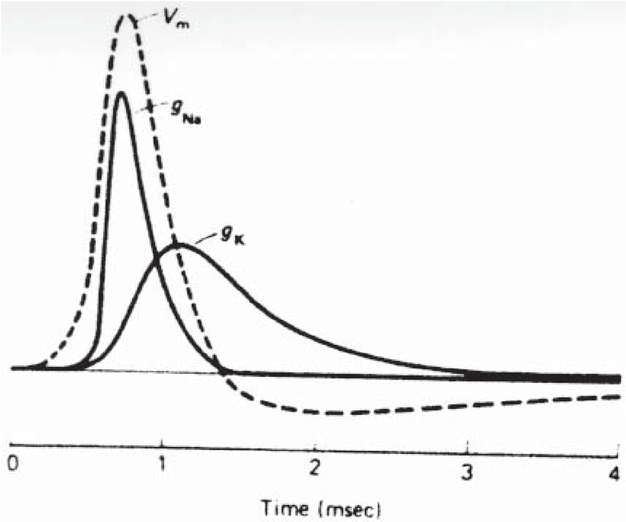


Fig. 9-4. The action potential results from the opening and closing of voltage gaited Na^+ and K^+ channels. Adapted with permission from Koester J. Voltage-Gated ion channels and the generation of the action potential. In: Kandel ER, Schwartz JH, Jessel TM, eds. *Principles of Neural Science.* 3rd ed. Norwalk, Conn: Appleton and Lange; 1991: 110.

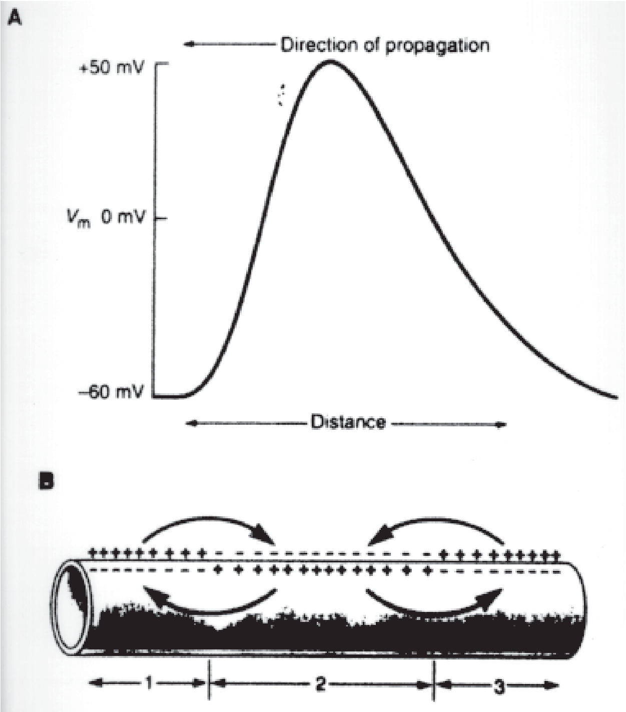


Fig. 9-5. Local changes in polarization (2) change the relative concentrations of ions across adjacent areas of the neuron (1,3). Depolarization will spread in both directions. During normal function however, diffusion of K^+ out of the region, which has already been depolarized (3), balances the local influence and the region does not depolarize or propagate the impulse in a retrograde direction. Adapted with permission from Koester J. Passive Membrane Properties of the Neuron. In: Kandel ER, Schwartz JH, Jessel TM, eds. *Principles of Neural Science.* 3rd ed. Norwalk, Conn: Appleton and Lange; 1991: 100.

allowing K^+ to rush out of the cell, reestablishing the resting polarized membrane potential.

The recorded action potential occurs at one point along the cell membrane (Figure 9-5). To propagate the impulse, depolarization of adjacent portions of the membrane is necessary. As the focus begins to depolarize, it becomes positive in relation to adjacent points along the membrane. The focus acts as an anion sink, "sucking" in the negative charges and causing the adjacent area to become less negative. Eventually, threshold is reached and the adjacent area then becomes the focus as it depolarizes.

The speed and efficiency with which impulses are propagated are radically different between unmyelinated and myelinated fibers. Depolarization proceeds longitudinally along the entire length of the axon in unmyelinated fibers. The segmental myelin sheaths found in myelinated fibers do not allow alteration in the ionic flux to occur. The impulse is propagated by jumping from node of Ranvier to a subsequent node, allowing for very rapid conduction velocities, upward of 70 m/s.

Axonal Transport

The protracted distance between the cell body and the distal reaches of the axon poses unique demands on the neuron. Energy driven systems have developed that maintain constant contact between the soma and the synapse. Three main modes of active transport along the axon have been recognized.

Fast Antegrade Transport

Constant utilization of neurotransmitters at the synaptic cleft and associated loss of cell membrane through pinocytosis requires rapid and constant replenishment. Materials, including neurotransmitter laden vesicles; mitochondria; and membrane components (eg, glycoproteins, glycolipids, lipids), travel along the fast antegrade transport system at a rate of 410 ± 50 mm/day. Ochs and Hollingsworth[13] exhibited the strong dependence of fast transport on oxidative metabolism. Fast transport came to a halt in nerves deprived of oxygen by placing them in a nitrogen environment for no more than 30 minutes.

Slow Antegrade Transport

Slow transport has been delineated into separate constituents, a and b.[14] Slow component a (SCa) is the slower of the two, moving at a rate of 1 to 3 mm/day. Neurofilament triplet proteins and tubulins are the principal materials transported by this system. Slow component b (SCb) travels more rapidly, 3 to 6 mm/day. Proteins such as actin, clathrin, and other microfilaments are carried by SCb. It has been suggested by Wujek and Lasek[15] that SCb is the rate-limiting step in axonal regeneration following nerve injury. The elements provided by SCb form the framework for the growth cone as well as the axon.

Retrograde Transport

Transport of materials from the periphery back to the cell body underlies three main functions: (1) depleted neurotransmitter vesicles are returned to the cell body and restored to full potency; (2) waste materials are cleared from the axonal fringes for degradation; and (3) it has also been postulated recently that trophic substances such as nerve growth factor (NGF) proceed proximally, providing the soma with information regarding the integrity of the cell. Deprivation of this trophic feedback is felt to be one factor heralding chromatolysis and the subsequent shift to regenerative processes during nerve injury.[16] The rate of retrograde transport varies from 100 to 220 mm/day.[17,18]

Transport Models

While the precise mechanisms of axonal transport have yet to be fully elucidated, several elegant theories have been developed. Taking into account the participation of microtubules, the utilization of ATP, and the need for Ca^{++}, Ochs[19] constructed the transport-filament hypothesis of fast axonal transport. Microtubules form a virtual track system along the length of the axon. A carrier protein binds the material to be transported to the microtubules. In a system reminiscent of the actin-myosin sliding filament complex utilized in muscle contraction, a racheting of the carrier proteins, driven by a Ca^{++}-Mg^{++} ATPase, allows the material to be passed from carrier protein to subsequent carrier protein. Fast antegrade transport employs kinesin as its transport protein, while a form of dynein is used for retrograde transport.[12]

In the past, slow antegrade transport was felt to reflect peristaltic waves inherent to the neuron. Ochs and Brimijan[16] suggest in their unitary hypothesis that the same system envisioned for fast antegrade transport may also be utilized in slow transport. Early detachment of tubulins and other structural proteins from the microfilament track might give the appearance of a slow moving wave.

Architecture of the Nerve

The trinary layers of supporting connective tissues, both individually and collectively, safeguard the neurons from mechanical and biological injury (Figure 9-6).

Fascicular Composition

The structural topography of the nerve trunks imparts a considerable defense against functional loss. Sunderland[20] reflects that at the root level, individual fibers destined to become specific distal branches are inextricably entwined with fibers from other virtual branches (Figure 9-7). As the fascicles extend peripherally, the fibers perpetually interweave, converging and diverging into new and different fascicular assemblies. The rate at which the confluence of fibers changes remains controversial, ranging from 1.5 to 2.5 cm.[7,9] Teleologically, it can be suggested that the plexiform configuration developed to enhance the tensile strength of the nerve trunk and help protect against mechanical deformation. The constantly changing position of individual fibers within the nerve also reduces the likelihood that a focal injury might damage a large

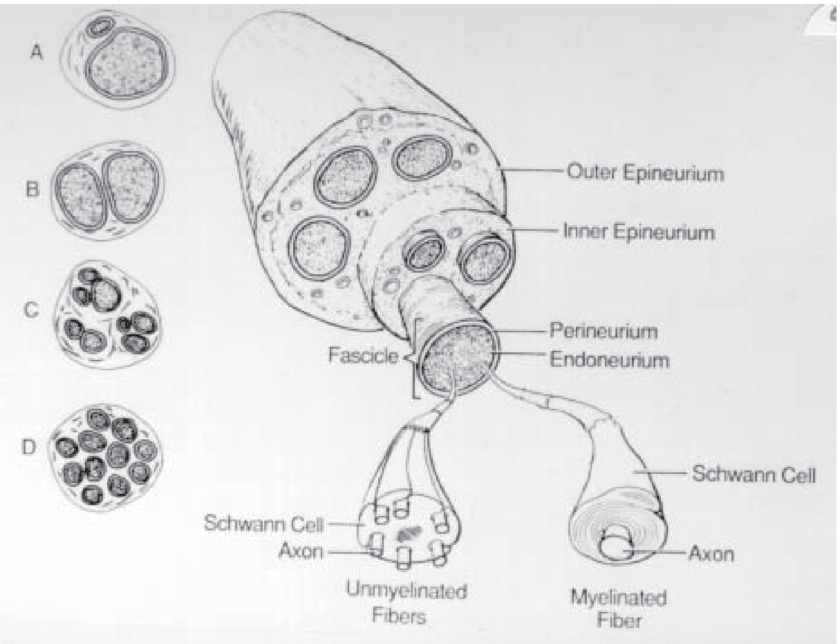


Fig. 9-6. Architecture of the peripheral nerve. Connective tissue elements include the epineurium, perineurium, and endoneurium. A mix of myelinated and unmyelinated fibers are commonly found within the same fascicle. **A**, **B**, **C**, and **D** show cross sections of fascicular composition at different levels. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure, Function and Reconstruction.* New York: Raven Press; 1990.

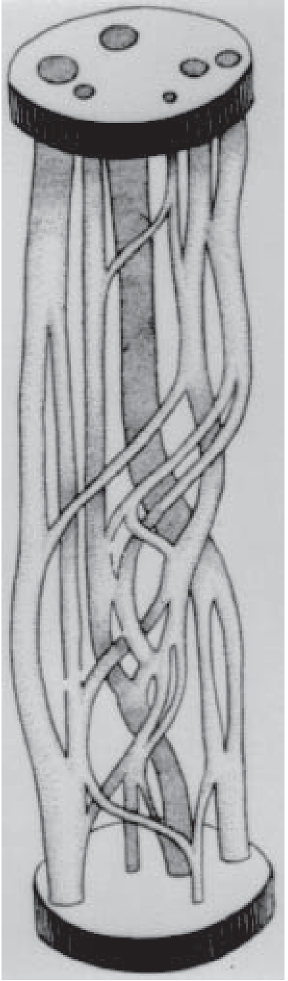


Fig. 9-7. Illustration of the interweaving plexus formations observed in a segment of the musculocutaneous nerve. Adapted with permission from Sunderland S. *Nerve and Nerve Injuries.* Edinburgh, Scotland: Churchill Livingstone; 1978: 32.

number of fibers destined to innervate a specific muscle group or sensory distribution.

Endoneurium. The neuronal complex of axon, Schwann cell, and basement membrane are surrounded by a loose meshwork of collagen fibers, termed the endoneurium. According to Sunderland,[20] the endoneurium imparts some protection to the neuron against stretch injury. Its capacity, however, pales in comparison to that provided by the perineurium.

Perineurium. The perineurium, the investing multiple layers of connective tissue surrounding distinct fascicles, is pivotal in maintaining the integrity and internal milieu of the nerve fibers. Two to five layers of flat, interdigitating cells linked by tight junctions form the blood-nerve barrier.[21] Large proteins, toxins, antigens, and infectious agents are prevented from entering the fascicles. Active transport of essential materials does occur across the blood-nerve barrier. The endoneurial fluid pressure is also maintained by the perineurium. Interdigitating between the lamellae are dense bundles of collagen and elastin fibers.[22] The perineurium is paramount in providing tensile strength and elasticity to the nerve. The response of the nerve to stretch has been examined extensively.[23–26] When a nerve is stretched, the gross undulation of the trunk is initially taken up. Further load leads to straightening of fascicular meandering. At the limits of elastic deformation, the epineurium ruptures. Further tension leads to plastic deformation of the perineurium, all efforts focusing on maintaining the integrity of the fascicles. The events following further loading remain controversial. According to Sunderland and Bradley,[26] the fluid nature of the individual neurons leads to disruption of some fibers prior to disruption of the perineurium. Haftek[24] alternatively suggests that elongation of the perineurium induces a tightening constriction around the fascicles, leading to myelin and eventually axonal injury. Rupture of fibers directly from the increased tension occurs at the perineurial level before the endoneurial level.

Epineurium. The most superficial layer is the epineurium. A loose areolar meshwork of collagen fibers, replete with lipid globules provides fundamental protection against compressive forces. Terzis and Smith divide this stratum into two components. The inner constituent surrounds each fascicle, providing interfasicular support and protection. The outer portion invests the entire nerve trunk.[7] The abundance of epineural tissue varies along the length of the nerve. Areas with larger numbers of fascicles generally possess a greater cross-sectional area of epineurium. For example, 88% of the sciatic nerve at the gluteal level is composed of epineural connective tissue, whereas, the epineurium comprises only 22% of the ulnar nerve at the medial epicondyle.[9] It is also noted that greater amounts of epineurium can be found at levels where nerves cross joints.

Vascular System of the Nerve

The peripheral nervous system is endowed with a multitiered vascular complex that preserves blood flow to the neurons even in the face of mechanical

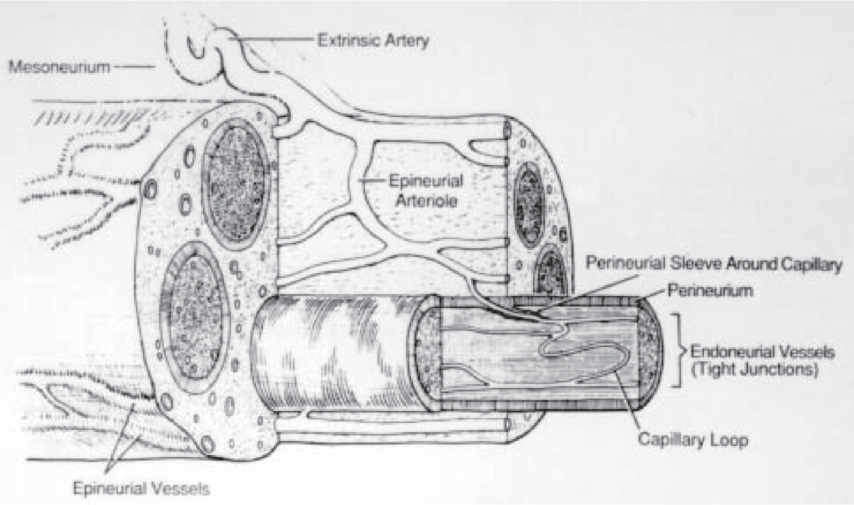


Fig. 9-8. The vascular system of the peripheral nerve. Vessels from periosteum and muscles pierce the epineurium. Branches enter the fascicles, feeding longitudinally aligned vessels beneath the perineurium. Vessels connecting the perineurial and endoneurial plexi follow an oblique path. Endoneurial capillaries form double U–loops, which permit rapid shift in blood flow to accommodate changes in position, pressure, temperature, or injury. Adapted with permission from Terzis JK, Smith KL. *The Peripheral Nerve: Structure, Function and Reconstruction.* New York: Raven Press; 1990.

disturbances. As previously discussed, a constant source of oxygen is necessary to sustain integral energy dependent systems such as axonal transport and impulse propagation. Over the past two decades, the salient works of Lundborg have expanded the current understanding of the neural vascular system.[25,27–29] Vascularization of peripheral nerves can be divided into extrinsic and intrinsic systems (Figure 9-8).

Extrinsic System

The extrinsic system incorporates branches emanating from local arteries in addition to muscular and periosteal vessels.[29] The myriad inputs permit continuous blood flow during movement (eg, translation across moving joints). The tortuosity of the vessels also allows for position changes as slack is taken up when the nerve becomes relatively taut. This rich blood supply becomes increasingly important during mobilization and reparative surgeries. Several studies have shown that significant transposition of several centimeters will ultimately not disrupt blood flow from the extrinsic system.[29,30] Branches from the extrinsic system pierce the epineurium and connect with the intrinsic system.

Intrinsic System

The intrinsic microcirculation incorporates three levels of anastomotic plexuses, each supplied by penetrating branches from more peripheral levels. Longitudinally aligned arterioles and venules make up the epineurial plexus. Vessels course around and between fascicles. Branches penetrate the outer layers of the perineurium, supplying flow to each fascicle. The perineurium provides significant protection to the numerous longitudinally oriented vessels, interdigitating between the lamellae and those at the endoneurial level. The ramifying vessels connecting the perineurial and endoneurial plexuses follow oblique courses through the innermost layer of the perineurium.[27] This configuration might play an important role during nerve injury. Lundborg suggests that in the face of rising endoneurial fluid pressure, focal ischemia may occur as these oblique vessels are compressed.[27] The endoneurial microcirculation consists of abundant capillary beds. Segmented, longitudinally arranged vessels are linked in a double U-loop formation.[29]

Interestingly, blood flow at any level of the intrinsic system responds to the subtle whims of external forces. Rapid shifts in the flow patterns arise following change in position, pressure, temperature, or injury. Thus, efficient utilization of available flow is ensured.[29]

Vascular Innervation

The sympathetic autonomic nervous system innervates the vasa nervosum. Strong stimulation of the sympathetic chain can lead to profound vasoconstriction at the neural level. It has been postulated that extended durations of increased sympathetic tone may lead to abnormal nerve function and may play a role in sympathetically maintained pain syndromes.[27]

PATHOPHYSIOLOGY OF NERVE INJURIES

Classification of Nerve Injuries

Profound insights into the pathophysiology and characterization of nerve injuries were undoubtedly spawned by the innumerable casualties sustained during World War II. Several systems were developed in an effort to clarify the seemingly heterogeneous clinical presentations of nerve injuries in continuity.

Seddon's Classification

Seddon[31] proposed the division of these injuries into three categories. Terms coined by Professor Henry Cohen in 1941 were adopted to describe the levels of injury: (1) neuropraxia, (2) axonotmesis, and (3) neurotmesis.

Neuropraxia. Neuropraxia (literally: nerve nonaction) is characterized by paralysis that occurs in the absence of distal degeneration.[31] Subsequent studies by Denny-Brown and Brenner[32] confirmed focal demyelination as the underlying pathology. Clinical observations at the time suggested that large myelinated fibers were more apt to be involved. Motor loss predominates, with diminution of vibratory and proprioceptive sensation, the most common sensory abnormalities. Less commonly, decreased touch sensation may occur and rarely is deep pain perception completely abolished. Paresthesias are very common and may be the harbinger of returning sensory function. In Seddon's study,[31] the most common etiology was an injury resulting from a compressive force. The time course from onset of injury to complete recovery of motor and sensory function ranged from 1 week to 6 months with 10 weeks as the average. Recovery in Sunderland's population of radial nerve injuries ranged from 17 to 60 days.[33] Recovery is spontaneous. Not uncommonly, several weeks of significant functional loss will be abruptly supplanted by exceedingly rapid recovery.

Axonotmesis. Axonotmesis (literally: axon cutting) suggests an injury to the nerve of such magnitude that distal degeneration occurs. The supporting structures: the epineurium, perineurium, endoneurium, and Schwann cells, however, remain intact. In the population studied by Seddon,[31] 43% of all axonotmetic nerve injuries occurred in the face of closed fractures, dislocations, or fracture/dislocations. Compressive injuries ranked a distant second at 18%. The manifestations of an axonotmetic injury can not clinically be differentiated from a more severe injury in the acute setting. Sensory, motor, and sudomotor functions of the involved axons are abolished. Over time, the excellent levels of recovery help to clarify the modesty of the lesion. Muscle and sensory return is not as rapid as that seen in neuropraxic injuries. The chronicity is directly proportional to the level of injury and can be approximated by dividing the distance to be traversed from the injury focus to the end organ by the rate of regeneration, estimated at 1.5 mm/d.[31]

Neurotmesis. The term neurotmesis (literally: nerve cutting) is something of a misnomer. While it implies total severance of the entire nerve, it was meant to describe an injury in which all functional elements have been severely damaged. The epineurium may in fact be intact at visual inspection. A remarkable 97% of Seddon's group sustaining this level of injury had experienced penetrating wounds, whether from gunshots, stabbings, or lacerations.[31] The prognosis for a neurotmetic lesion is invariably poor. Spontaneous recovery is inimitable. Surgical coaptation may lead to functional return, although resolution of deficits is rarely complete.

Sunderland's Classification

Seddon's classification of nerve injuries was a bold initiative, enhancing the ability to discern clinically salient degrees of injury. Several ambiguities in Seddon's monograph spurred others to develop more elaborate classification systems. While complaints regarding the use of obscure Greek derivatives to describe the different levels seem petty at this time, more significant shortcomings were well founded. Seddon's definition of axonotmesis for example, was specific. It asserted the integrity of the epineurium, perineurium, endoneurium, and Schwann cells. His description of the clinical presentation, however, did not match the presumed anatomical distortion. It was suggested by Sunderland that in cases where more extensive injuries have been sustained (eg, a fusiform neuroma),[31] the strictly defined category of axonotmesis was not truly correct.[34] More precise predictions of functional return, the timing of surgical exploration, and potential microsurgical reconstruction warrant the ability to determine more accurately the severity of an injury. Sunderland's classification system

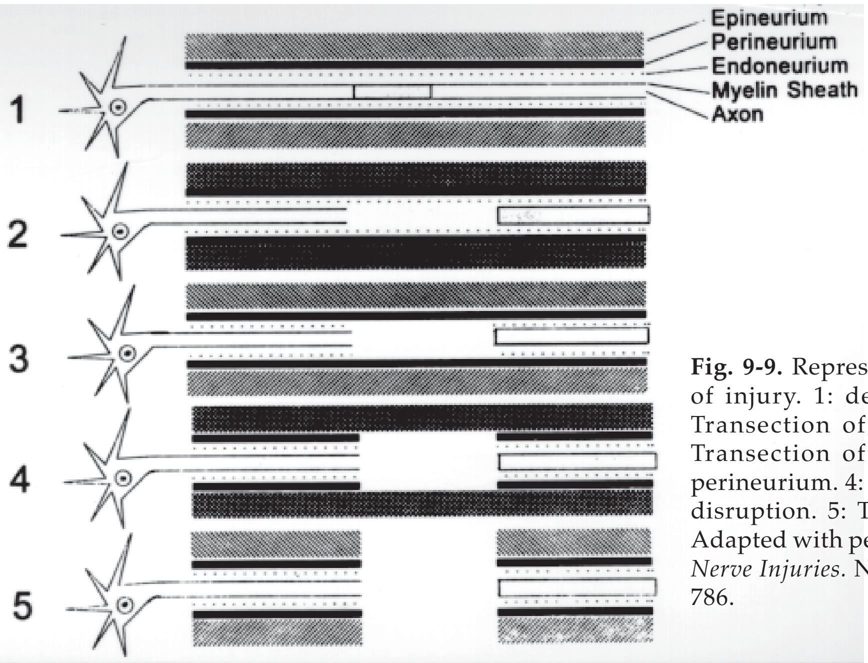


Fig. 9-9. Representation of Sunderland's five degrees of injury. 1: demyelination (conduction block). 2: Transection of axon with intact endoneurium. 3: Transection of the axon and sheath inside intact perineurium. 4: Transection of perineurium, fascicular disruption. 5: Transection of the entire nerve trunk. Adapted with permission from Sunderland S. *Nerve and Nerve Injuries.* New York: Churchill Livingstone; 1978: 786.

was based on detailed anatomical investigations of peripheral nerves and nerve injuries. Five degrees of injury were defined (Figure 9-9).

First-degree injury. A first-degree injury is synonymous with neuropraxia. Focal conduction block is caused by mechanical disruption of the myelin sheath. The axon and all supporting structures remain intact. Wallerian degeneration does not occur. Recovery is rapid and spontaneous.

Second-degree injury. A second degree injury correlates with Seddon's strict definition of axonotmesis. The axon is damaged, but all supporting structures, including the endoneurium and Schwann cell tubes, escape injury. Wallerian degeneration of the distal stump begins expeditiously following the trauma. As the architectural pathways remain unharmed, the regenerating axon has an excellent chance of growing down the proper endoneurial tube, innervating the correct end organ. Recovery is influenced by the level of injury and the rate of advancement of the growth cone. The Hoffman-Tinel sign, the provocation of tingling or lancenating sensations caused by tapping superficially on the sensitive region of a nerve, may be a useful tool providing a gross estimate of the progress and level of axon tip expansion.

Third-degree injury. Intrafascicular derangement typifies a third-degree injury. Within the uninjured perineurium, axonal and endoneurial degeneration transpires. Several potentially confounding obstacles may hamper regeneration. Endoneurial edema might result from alterations in vascular permeability following the insult. The lack of lymphatic channels and the perineurial blood-nerve barrier prevent adequate drainage from the intrafascicular space.[35] Direct injury to the endoneurial capillary plexus or obliquely situated feeder vessels may result in frank hemorrhage. Vascular stasis, ischemia, and, subsequently, fibrosis may block further recovery.

The loss of endoneurial tube continuity leads to a virtual free-for-all, as regenerating axon tips extend randomly toward endoneurial tubes at the distal side of the injury focus. An axon may meet with one of several fates. It may (*a*) pass into the correct distal endoneurial tube and innervate the proper end organ; (*b*) enter the wrong distal tube with ineffectual results; (*c*) axonal sprouting may lead to an axon-endoneurial tube mismatch, with too few tubes available to accommodate all the axons; and, again, the end organ may never be correctly innervated; or (*d*) fibrosis and organizing hemorrhage may entangle the axon, preventing further progression, and local sprouting will result in neuroma formation. The level of injury also has a profound effect on potential functional return. As previously discussed, fibers destined to become specific branches are intermingled at very proximal levels. Therefore, axons injured at high levels are less likely to regain proper end organ contact. As more distinct branches form distally, the likelihood of successful reinnervation improves. In situations where similar muscle groups are erroneously innervated by the wrong axons, neuromuscular reeducation may be extremely beneficial in improving functional recovery.

As would be expected, the onset of recovery is more delayed than in second degree injuries. Commonly, recovery is incomplete with persistent sensory and motor deficits that are variable in regard to severity.

Fourth-degree injury. Perineurial disruption, in addition to the more internal structures, is the hallmark of a fourth-degree injury. Rarely is any useful function obtained by spontaneous recovery. Neuroma formation is more the rule than the exception. Excision of the affected segment and surgical repair are invariably necessary for any hope of functional return.

Fifth-degree injury. A fifth-degree injury implies complete disruption of the entire nerve trunk, including the epineurium. Ironically, an injury of this magnitude may in fact lead to a better outcome surgically than a fourth-degree lesion, depending on the mechanism of injury. A sharp laceration with very isolated destruction responds well to early surgical intervention. Outcomes following crush or stretch injuries parallel those seen in fourth-degree injuries and are treated similarly.

Utility of Classification Systems

Classifications of peripheral nerve injuries have helped define the severity of injuries, have led to more precise prognostic evaluations, and have guided clinicians toward more rational and appropriate treatment plans. It should be noted, however, that pure lesions, in which all fibers sustain the same degree of injury, are not common. Of 537 nerve lesions in continuity evaluated by Seddon,[31] only 22% were pure. Thus, while gross estimates may be made in regard to recovery, the distribution of injury intensity across the inter- and intrafascicular levels adds another variable that may impact significantly on the return of sensory, motor, or sympathetic function.

END ORGAN CHANGES FOLLOWING DENERVATION

Wallerian Degeneration

Nerve injuries of magnitudes great enough to disrupt the integrity of the axon (ie, second degree or more severe) invariably result in stereotypical degeneration of nerve fibers distal to the focus of injury. This orderly progression was first characterized by Waller in 1852[36] and has been eponymously termed "Wallerian degeneration." The physiologic changes occurring can be divided into those involving the axon, Schwann cells, myelin sheath, and the macrophage response. Wallerian degeneration is erroneously portrayed as merely a destructive process. It is truly an elegant system, initiating the complete destruction of nonviable axonal components, removing all waste products, and establishing a fertile environment that may successfully guide forthcoming regenerating axons (Figure 9-10).

Axon Changes

The earliest axon changes are those seen at the terminal end of the nerve, in motor neurons at the neuromuscular junction. Miledi and Slater[37] demonstrated that an electrical impulse could be propagated along the distal portion of a transected rat phrenic nerve for upward of 8 hours. A rapid decline ensued at this point with complete loss of the ability to evoke an action potential over the next several hours. A strong direct correlation between loss of impulse propagation and cessation of spontaneous end-plate potentials was observed. Histologic changes paralleled those seen physiologically. Within 3 to 5 hours following the loss of spontaneous end-plate potentials, the normal structure of the end plate was severely distorted. Mitochondria became swollen and spherical in shape. Disorganization and eventual fragmentation of the cristae ensued. The axoplasm and its resident organelles disintegrated into amorphous clumps, ultimately engulfed by Schwann cells. Eventually, all that remained was a watery cytoplasm, devoid of functional elements. Schwann cells protruded into the synaptic cleft, completely enveloping the terminal end of the axon, disrupting contact between the nerve and muscle at the neuromuscular junction.[38]

Granular disintegration of the axon rapidly follows failure at the terminal junction. Collapse of neurofilaments and microtubules, which impart structural integrity to the axon, along with diminution of axoplasmic volume, are the cardinal events marking the onset of axonal degeneration.[39] Striking changes within the cell body and proximal stump are also evident within days and peak over the subsequent days to weeks. In motor neurons, the nuclei become eccentric and migrate to the pole opposite the axon hillock. Disappearance of Nissl bodies is noted as well as disintegration of the rough endoplasmic reticulum.[40] There is a virtual halt to

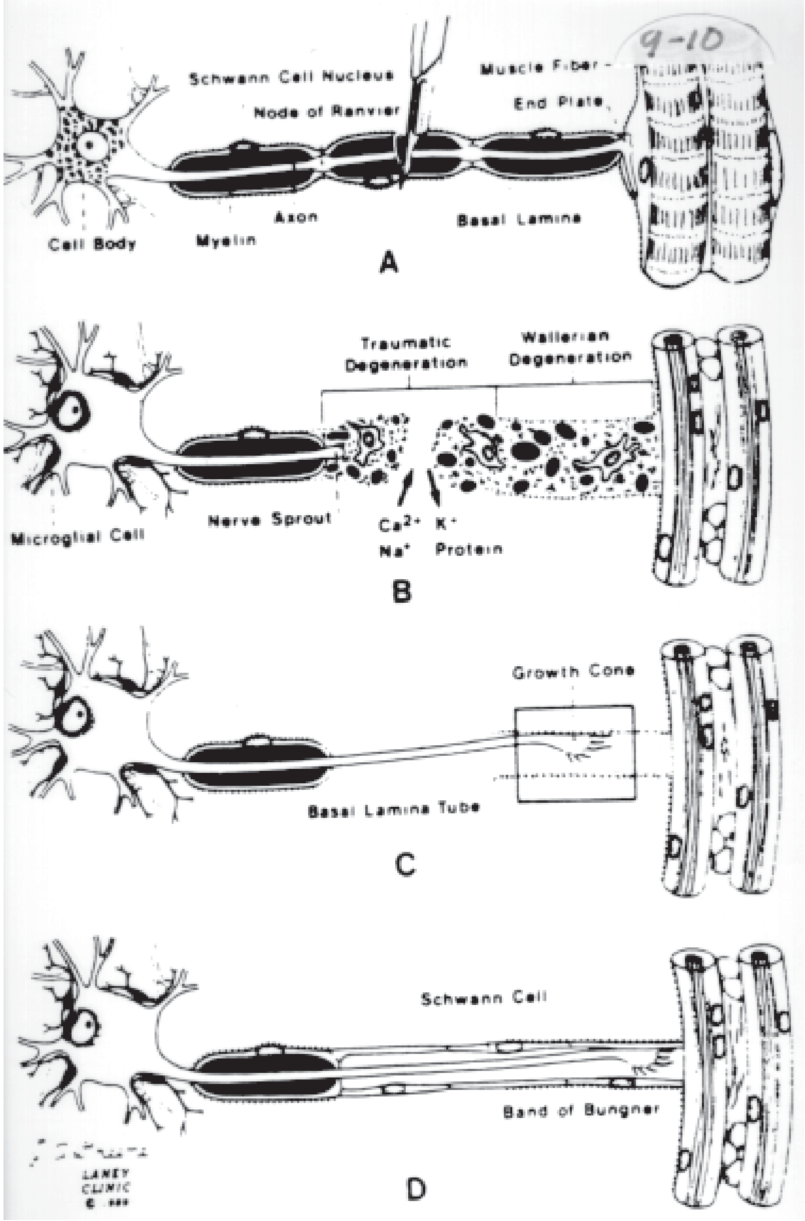

9-10
Schwann Cell Nucleus
Node of Ranvier
Muscle Fiber
End Plate
Cell Body
Myelin
Axon
Basal Lamina
A
Traumatic Degeneration
Wallerian Degeneration
Microglial Cell
Nerve Sprout
Ca^{2+} K^+
Na^+ Protein
B
Growth Cone
Basal Lamina Tube
C
Schwann Cell
Band of Bungner
LAHEY CLINIC
D

Fig. 9-10. Peripheral nerve degeneration and regeneration. **A**: Injury to normal fiber. **B**: Wallerian degeneration of distal axon. Basal lamina tube remains intact distal to the injury focus. **C**: Growth cone is formed and is attracted by chemotactic agents. **D**: Schwann cell line the basal lamina, attracting axons and eventually laminating the axon in myelin. Adapted with permission from Seckel BR. Enhancement of peripheral nerve regeneration. *Muscle Nerve.* 1990;13:785–800.

the production of neurotransmitter and a down regulation of messenger ribonucleic acid (mRNA).[41] Production of proteins crucial to anticipated regeneration, such as actin and tubulin, which are integral parts of the growth cone, are upregulated[42] while the initial production of structural neurofilament and microtubule proteins is diminished. The cross-sectional area of nerve fibers also decreases and may reflect this loss of neurofilament protein production.[43]

Initiation of Wallerian Degeneration

Debate has raged over the past 150 years as to what triggers the onset of axonal degeneration. Waller summized intuitively that a loss of trophic support arises following the severance of the axon from its supporting cell body.[44] While the failure of nutritive support may partially explain the onset of chromatolysis and axon disintegration, the rapidity with which degeneration occurs is characteristic of an active, destructive process.[45] Others have suggested that the loss of impulses traveling down the nerve might lead to axonal atrophy, analogous to disuse atrophy seen in immobilized muscle.[46,47] This theory has been recently refuted by several studies in which electrical stimulation mimicking the firing pattern of motoneurons failed to retard axonal degeneration in axotomized nerves in rabbits.[43,48] Gutmann and Holubar[44] suggested an autolytic process as the likely cause of degeneration. Current studies examining ionic fluctuations during axotomy support this hypothesis.

LoPachin et al[49] showed that shifts in the concentration of specific ions precede even the earliest morphologic transformations. The changes in intraaxonal ionic composition follow a sequential pattern. In rat sciatic nerves, loss of K^+ and Cl^- is noted initially at the 8-hour mark. Intracellular levels of Na^+ and Ca^{++} begin to rise rapidly between 16 and 48 hours postinjury. Progressive deactivation of the Na^+-K^+ ATPase pump and potentially an active Ca^{++} pump[39] secondary to the loss of energy input are possible causes. As previously discussed, without the function of the Na^+-K^+ ATPase, K^+ escapes from the cell, along its concentration gradient. The relative impermeability of the neuronal membrane to Na^+ and Ca^{++} accounts for the time delay. As Na^+ rushes into the cell, the gated ion channels for both Na^+ and Ca^{++} become increasingly permeable, allowing for an even greater influx of Na^+ and Ca^{++} ions. The Ca^{++} influx activates endogenous proteases which initiate axonal destruction.

Another school of thought turns the antegrade trophic support theory on its end. Initially, following axotomy, there is down-regulation of neurofilament gene expression. It has been suggested[50,51] that substances akin to NGF may permit specification of the level of neurofilament gene expression at the cell body. The retrograde transport of this factor from the periphery is dependent on intact interactions between the nerve ending and specific target or accessory cells. Thus, the loss of peripheral retrograde feedback might trigger the shift from production of proteins needed to maintain regular neural function and integrity to that necessary for reconstruction of the damaged axon following clearance of axonal debris.

Schwann Cell and Myelin Sheath Changes

Within 2 days following axotomy, profound changes in myelin composition occur. Production of mRNA, sequenced for the production of myelin proteins, is markedly reduced. The internodal segments collapse and become disorganized. Local Schwann cells extend cytoplasmic phalanges through the myelin sheaths, fragmenting them into myelin ovoids. In rats, the ovoids are progressively destroyed over the ensuing several weeks, culminating in disorganized lipid whorls.[45] These Schwann cells, occupying the space previously filled by the internodal myelin are termed bands of Bungner. A proliferation of Schwann cells occurs, providing the groundwork and guidance system for future axonal regeneration. In sensory and sympathetic neurons, the production of NGF as well as NGF receptors has been localized to Schwann cells forming bands of Bungner. Interestingly, it was observed[52] that disruption of axon-Schwann cell contact triggers the expression of NGF receptors of the surfaces of the proliferating Schwann cells.

Role of Macrophages

The mechanism underlying the removal of axonal and myelin debris has been a focus of contention for many decades. In the 1920s, Ramon y Cajal and Swanson[53] described the synergistic roles played by Schwann cells and macrophages. It is only recently, however, that his cooperative paradigm has been confirmed experimentally. Stoll and colleagues[54] observed Schwann cells interrupting, phagocytizing, and degrading myelin sheaths during the earliest hours following axotomy. It was proposed that Schwann cells likely initiate degradation of their own myelin sheaths.[54] They do not, however, participate in the removal of myelin breakdown products. Elimination of myelin is accomplished by macrophages. A vast number of macrophages are chemotactically attracted to the degenerating neuron from the circulation. A paucity of resident macrophages also assist in the clearance. Beuche and Friede's[55] cogent study revealed that when nerve fibers degenerate in an environment devoid of macrophages, collapsed myelin sheaths persist for many weeks. The evacuation of debris is not the only function of macrophages. Synthesis and release of the cytokine interleukin-1 by macrophages is essential for the production of NGF by Schwann cells responding to nerve injury.[56]

Reinnervation

The degenerative phase produces a milieu conducive for regeneration. Cell body protein synthesis has switched to the production of actin and tubulin, essential to the construction of the growth cone. First described by Ramon y Cajal and Swanson,[53] the growth cone is a specialized conical swelling that develops at the distal end of the proximal stump. The growth cone has been described as ameoba-like,[53] extending lamellipodia which facilitate movement toward the endoneurial tube across the focus of injury into the distal stump. Growth cone movement and axonal elongation require several guiding elements. Endoneurial tubes in the distal stump persist following the removal of myelin and axonal debris. As previously mentioned, the disruption of contact between axons and Schwann cells triggers a rapid proliferation of the latter, which line the basal lamina. These Schwann cells produce receptors for growth-associated proteins such as NGF and growth-associated protein-43 (GAP-43), which serve as chemoattractants for the growth cone.[52,57,58] The growth cone follows the weak external chemotactic gradient, which is amplified internally.[59] It has also been shown that there may be some selectivity, improving the likelihood that certain types of neurons will enter matching endoneurial tubes. Contact of sensory growth cones with sympathetic neurons causes a collapse of the cone and redirection of its growth.[60]

The opportunity for axon growth into suitable distal tubes is somewhat self-limited. Schwann cell mitosis will continue only if contact with a growing axon is made.[61] When contact is not established between an extending nerve fiber and Schwann cells within the distal endoneurial tube, the diameter of the tube shrinks to 10% to 20% of its original size, hampering subsequent attempts at reinnervation.[62]

Muscle Changes Following Denervation

Changes in muscle function and morphology following denervation are congruent with those occurring in the nerve fiber. Loss of innervation triggers drastic transfiguration at the nuclear level, which leads to alterations in membrane composition, ionic channels, and neurotransmitter receptors. Macroscopic transformation of muscle tissue constituency and fiber organization ensue. While manifestations of denervation are commonly construed as deleterious, many enhance the tissues' responsiveness to reinnervation. External factors, including the extent and chronicity of nerve injury, the association of concomitant injuries, and age may have more to do with the ultimate functional outcome than the changes caused by denervation within the muscle itself.

Muscle Fiber Changes

Histological findings. The most characteristic early change in denervated muscle fiber is disruption of sarcolemmal nuclei, which become rounded and hyperchromatic and may become internalized within the myofiber.[63] A gradual reduction in fiber caliber proceeds. While subtle differences exist, the rate of extrafusal muscle fiber atrophy remains uniform across several mammalian species.[64–66] Bowden and Gutmann[65] observed no evidence of degeneration of muscle fibers during the first 3 months following denervation. The internal integrity of the sarcomeres remained intact. The cross-sectional area of muscle fibers decreased precipitously over the initial 60 to 90 days and then stabilized. These findings are consistent with Sunderland's data[9] in opossum and Gutmann and Young's assessment[64] in rabbits, which suggest that atrophy levels off at 70% to 90% by the third month.

Electron microscopy and biochemical studies have revealed that loss of fiber size reflects a loss of myofilaments. Early on, filament disappearance is sporadic and is observed only at the ends of the myofibers. The loss of myosin heavy chains precedes the loss of actin light filaments. By 4 to 6 months, some disorganization of the hexagonal filament arrangement occurs. At this point, changes are readily reversible with reinnervation. Interestingly, a difference in the rate of myosin heavy chain loss has been observed between fast, glycolytic, and slow oxidative muscle fibers. Fast fibers maintain myosin integrity for 4 to 5 times longer than slow fibers.[66] It has been suggested that the firing frequency ratios of the fibers may underlie the difference. Lack of stimulation is unusual in slow fibers, which are recruited early and commonly produce tonic, low-level activity. Thus, they may be more sensitive to the loss of innervation. Fast fibers, alternatively, are recruited late for brief bursts of speed and power and may be more resistant to a paucity of stimulation.[67]

A decrease in mitochondrial size and number, an increase in lysosomes, and a progressive depletion of oxidative and glycolytic enzymes closely follows the loss of myofibrils.[63] Again, these changes are late and not universally discernible for several months. Bowden and Gutmann[65] presumed that deterioration of fibers is incredibly deliberate. It may be upward of 3 years or more before a level of decay is achieved that might confound attempts at reinnervation. Anecdotal reports have shown functional recovery following reinnervation of fibers denervated for 22 years.[68] It should be understood, however, that while the extrafusal fibers maintain their integrity in the face of atrophy for some time, other forces are at work that may compromise the feasibility of reinnervation as time goes on.

Muscle fiber atrophy. Muscle atrophy following denervation is the result of a combination of factors. Muller[69] observed a decrement of strength of 5% per day from initial levels, slowing to a loss of 25% at 7 days of immobilization. Protracted studies assessing strength loss during immobilization over several weeks revealed a leveling off of strength loss. The overall average at the end of 6 weeks was 8% per week with an overall loss of 48% of baseline strength.[70] Loss of contact between nerve and muscle deprives the end organ of trophic substances normally secreted by the motor neuron.[71] Davis and Kiernan[72] formulated two studies to determine relative responsibility for muscle atrophy. The extensor digitorum longus muscles in one group of Wistar rats were immobilized for seven days. Cross-sectional area decreased by 22%, consistent with the effects of immobilization described by other investigators.[69,73,74] In a group in which the muscles were both immobilized and denervated, cross-sectional area decreased by 35%. In a third group in which muscles were immobilized, denervated but injected with extracts of sciatic nerve, cross-sectional area reductions similar to those seen during pure immobilization were achieved. It was determined that approximately 60% of atrophy following denervation can be attributed to disuse, while the loss of neurotrophic influence accounts for the remaining 40%.[75] The importance of this delineation cannot be overstated. While much research is underway to develop techniques to augment reinnervation and the search to isolate neurotrophic substances continues, no clinically significant applications are currently available. Rehabilitative techniques that focus on limiting or overcoming disuse atrophy, however, are well founded and effective methods to enhance functional restoration.

Connective Tissue Changes

While loss of innervation leads to muscle fiber atrophy, collagen biosynthesis is unleashed and flourishes in the absence of neural inhibition. Sherman[76] observed the development of myogenic contractures in immobilized, denervated muscles within 4 to 6 weeks following injury. Myogenic contractures result from fibrosis involving the endomysium and perimysium. Little evidence suggests involvement of the epimysium. Types I and III collagen synthesis markedly increase following denervation. The collagen fibers become coarse, thickened, and enmeshed by the interweaving of cross-linking proteins. The ordinarily loose collagen meshwork surrounding myofibers tightens, mummifying atrophic muscle cells.[77] The effects of disuse, in contrast to muscle fibers, plays no role in the alteration of collagen synthesis in the face of denervation. Savolainen and colleagues[78] showed marked decreases in levels of collagen biosynthetic enzyme activity when muscles were merely immobilized. Denervated muscles, contrarily, expressed biosynthetic enzyme activity two and three times normal following neurectomy. The findings suggest that normal collagen synthesis adapts to the level of muscular activity and is under neural regulation. These findings have recently been confirmed by Virtanen and colleagues,[79] who not only replicated Savolainen's data, but also observed that reinnervation returned collagen biosynthesis to normal levels.

Denervation Supersensitivity

Supersensitivity or membrane instability, as it is termed in electrodiagnostic circles, is a hallmark of denervation. Two processes, the development of ectopic acetylcholine receptors and alterations in the electrical property of the sarcolemma are implicit in its development.

Acetylcholine receptor proliferation. In normal muscle, only the endplate, the region intimately associated with the presynaptic motor neuron bouton, possesses receptors for acetylcholine. A concentration of 3×10^7 receptors is found for every square micrometer. The inherent stability of the acetylcholine receptors is dependent on the influence of the motor neuron. Approximately 20% of receptors turn over rapidly, in less than 72 hours. The majority have half-lives of 6 to 13 days.[80] It is suggested that the rapidly decaying receptors are precursors to the more stable and mature receptors. Following propagation of an action potential, quanta of acetylcholine are released across the synaptic cleft and activate acetylcholine receptor channels at the endplate. The acetylcholine channels are somewhat generic and allow the passage of Na^+, K^+, and Ca^{++} cations. A net influx of Na^+ ions occurs that decreases the polarity of the cell, causing an endplate potential. The acetylcholine channels work similarly to an automobile starter and are not the direct generators of the muscle action potential. The decreasing negativity caused by the influx at the acetylcholine channel triggers voltage-sensitive Na^+ channels outside the endplate, leading to amplification of depolarization and, ultimately, propagation of the action potential along the length of the muscle fibers.[81]

Denervation leads to profound alterations in muscle cell function. The loss of neurotrophic support induces, at the nuclear level, the production of proteins destined to be extrajunctional acetylcholine receptors. Initially, the extrajunctional acetylcholine receptors form in clusters across the entire membrane. Their density increases, although only to one tenth of that established at the endplate. The entire cell membrane becomes exquisitely sensitive to acetylcholine. During the hiatus between denervation and reinnervation, these extrajunctional acetylcholine receptors remain immature and rapidly turn over every 6 to 35 hours. In contrast to junctional receptors, the extrajunctional receptors are not exposed to acetylcholine esterase.[82]

Alteration of membrane potential. The second major factor promoting the development of denervation supersensitivity is the alteration of the electrical properties of the sarcolemma. The increase in acetylcholine receptors increases the likelihood that even minimal stimulation may lead to depolarization. Changes in the ion channels also play a role. While there is no change in the conductance of Ca^{++} activated K^+ channels, a 60% reduction in the number of these channels has been observed.[83] Alterations in Na^+ conducting channels and a decrease in the activity of the Na^+-K^+ pump also develop.[38] The increased permeability to Na^+ influx and decreased K^+ efflux precipitates a decrease in membrane polarization from –70 to –55 mV. Thus, the muscle fiber teeters perpetually on the brink of depolarization.

Influence on Regenerating Nerve Fibers

During reinnervation, constant feedback between the new nerve fibers and the extrajunctional acetylcholine receptor clusters lead to the development of stable, mature neuromuscular junctions. While the protein has yet to be isolated, several studies suggest that skeletal muscle itself exudes a trophic factor that stimulates and attracts sprouting neurons.[84,85] As many as 4 to 5 neurons may be observed in association with a single muscle fiber during the rudimentary stages of reinnervation. Contact between nerve fibers and extrajunctional acetylcholine receptors leads to the evocation of endplate potentials. Despite their low quantal content, the lack of acetylcholine esterase and the hypopolarization of the cell membrane enable muscle activity.

Repetitive and increasing levels of stimulation to inhibit the synthesis of acetylcholine receptors and eventually only the area of the fiber in contact with the motor neuron retains receptors. The new connections enhance the stability of these receptors and allow them to mature. They also trigger the formation of acetylcholine esterase binding sites within the basal lamina of the synaptic cleft.[86] The trophic influence of the muscle on sprouting nerve fibers eventually wanes. As strong neuromuscular junctions are established and the regions of acetylcholine receptors become constricted and defined, the reinnervated motor fibers will not accept additional innervation from another nerve.[87] Ultimately, a normal relationship between motor neuron and muscle fiber is completed.

MECHANISMS OF NERVE INJURY

Compression Injuries

Compression of peripheral nerves is one of the most common types of injury, exemplified by entrapment neuropathies. Compression may be caused by edematous changes in a nerve as it passes through a narrow space. Alternatively, a normally capacious tunnel may be compromised by soft tissue swelling, fibrosis, fractures, or foreign bodies. Blunt trauma such as that seen in Saturday night palsy is also a common etiology. Despite such familiarity, compression injuries represent complex lesions.

Pathophysiology

The mechanism by which compression disrupts neural function has been intensely studied. Over the past 50 years, somewhat polarized theories have gained popularity ranging from predominantly ischemic injury to pure mechanical deformation. Presently, more integrated explanations have materialized.

Clinical descriptions of motor paralysis with variable sensory deficits, no distal atrophy, and rapid excellent recovery were made by Frazier and Silbert during World War I.[88] Seddon,[31] too, observed a similar clinical pattern, which he termed neuropraxia. It was not until 1944, however, that Denny-Brown and Brenner[89] experimentally confirmed Seddon's clinical hypothesis regarding pathologic changes in nerve fibers. Compression of cat sciatic nerve by either tourniquet or spring clip resulted in similar damage. Initially, edema was observed at the nodes of Ranvier with "powdering" of the myelin sheath. By 7 days after pressure application, the myelin had receded from the nodes closest to the edge of the cuff or clip. Thus, focal demyelination was confirmed.[32,89] Large myelinated fibers were more commonly injured than smaller fibers. Clinically, voluntary motor function had been abolished by pressures of 300 mm Hg applied for two hours. Motor excitability below the level of the lesion was 75% to 100% intact, while that above the level of the lesion had dropped to 0% to 5%. Denny-Brown and Brenner[89] suggested that segmental demyelination was related to ischemia and was not a consequence of direct pressure on the nerve fibers.

Ochoa and colleagues[90] conceded that mild compression, producing a metabolic conduction block that reverses rapidly with release of pressure, is caused by local ischemia. Conduction block lasting weeks to months without significant axonal degeneration was felt to be caused by direct damage to the nerve fibers. The characteristic lesion observed on teased fiber examination was displacement of nodes of Ranvier with invagination of one paranode by an adjacent one. Compression causes movement of the axon and myelin sheath away from the edges of the cuff, along the plane parallel to the nerve. The Schwann cell and basement membrane hold steadfast. A shear force is generated, dissociating the myelin from its parent Schwann cell. The invaginated myelin, torn from its nutrient source, degenerates. A focal barren area, void of myelin, develops.[90]

The findings of Ochoa and colleagues resulted from very high pressures (1,000 mm Hg) applied for several hours across a short segment (5.5 cm). Others employing lower pressures, which more closely resemble clinical situations, have not been able to reproduce these findings.[91] It is likely that a combination of primary mechanical deformation and secondary microvascular damage with edema and impaired local microcirculation produce the common clinical picture of conduction block.

Compression at levels as low as 30 mm Hg have been shown to slow venular flow.[35] Pressures upward of 80 mm Hg can completely arrest endoneurial circulation.[92] Reduction in venous flow further encumbers a normally sluggish endoneurial fluid drainage system. Disruption of oblique penetrating vessels may lead to increased vascular permeability and compromise of the blood-nerve barrier. Endoneurial blood flow, measured by laser doppler, has been observed to decrease by 50% in regions solely displaying focal demyelination.[93]

Axonal transport, which is exquisitely sensitive to ischemia, is disturbed at pressures as low as 20 mm Hg when sustained for upwards of 8 hours. At this level, mechanical distortion of the axon is unlikely. Distal supplies of cytoskeletal proteins, essential for axonal integrity and neurotransmitters utilized in synaptic transmission, may be jeopardized, crippling nerve function. Retrograde transport is also affected, limiting the flow of trophic factors from the periphery back to the cell body and undermining cell homeostasis.[94]

Clinical Considerations

Several studies have addressed the relative significance of ischemia and mechanical deformation in clinical settings. Gelberman et al[95] observed infraretinacular pressures of 32 mm Hg in patients with symptoms suggestive of carpal tunnel syndrome (CTS). Control subjects, without symptoms of CTS, had canal pressures averaging 2.5 mm Hg. This critical pressure was reproduced by Lundborg et al.[96] At 30 mm Hg, abnormal sensations such as numbness and parasthesias were observed 10 to 30 minutes following onset of compression. A decrease in sensory nerve action potential (SNAP) amplitude occurred concurrently. The amplitude decrement was not significant by current electrodiagnostic standards (ie, the amplitude was not decreased by greater than 50% when compared to the baseline amplitude). Gelberman and colleagues[97] noted at 40 mm Hg, decrements in sensory amplitudes of 40% within 10 to 30 minutes. Latencies were minimally delayed. Light pressure and two-point discrimination were not abnormal at this level and only after 4 hours of compression were vibratory sensibility abnormalities, tested at 256 Hz, observed. At 50 mm Hg, a significant drop in SNAP amplitude (ie, greater than 50%) was first observed within 15 minutes. Complete conduction block was achieved within 20 minutes. Motor latency delays were significant within 30 minutes. Abnormal sensibility to vibration and light pressure were documented at 20 minutes. Two-point discrimination measurement expanded beyond four mm at 30 minutes but was not completely lost until all sensation had been completely ablated. Motor strength faded from normal to fair over several hours. As with lesser levels of pressure, all parameters returned to normal abruptly after pressure release at 240 minutes. Pressures of 60 to 70 mm Hg produced findings similar to those found at 50 mm Hg.[97] Pressures of 90 mm Hg also produced reversible signs and symptoms when maintained for 60 minutes.[96] An animal study[98] employing rabbit nerves compressed at 80 mm Hg produced corresponding amplitude drops; however, maintenance of pressure for greater than 2 hours led to persistent diminution of action potential amplitudes.

A number of important conclusions regarding nerve tissue pressure thresholds can be made from these experiments. Pressure between 30 and 50 mm Hg may produce symptoms including numbness and parasthesias but reveal no objective signs aside from nonsignificant SNAP amplitude drops. This likely reflects mild local ischemic metabolic conduction block and may explain the 20% of patients with symptoms consistent with CTS but normal electrodiagnostic testing results.[99] Pressures greater than 50 mm Hg produce signs reflecting alteration in neurophysiologic function. Symptoms and signs manifest during short durations of pressures less than 80 mm Hg readily reverse with resolution of compression. Higher pressure or longer duration or both may lead to persistent findings, caused not only by ischemia but also by mechanical deformation and focal segmental demyelination. This level of injury is best exemplified by abrupt, focused application of high pressure such as that seen in compression of the radial nerve against the humerus during near-blast injuries, or alternatively, prolonged moderate pressure caused by entrapment of the radial nerve between the humerus and an external force as seen in Saturday night palsy. In these situations, where primarily segmental demyelination has occurred from mechanical deformation, recovery can take an average of 6 to 10 weeks and reflects the period of time required for remyelination.[100] Conduction block lasting greater than 10 weeks without evidence of Wallerian degeneration has been attributed to persistent endoneurial edema and elevated endoneurial fluid pressure. More severe compression may lead to not only demyelination but concomitant axonal injury.

Fascicular anatomy also plays a crucial role in the clinical manifestations of compression injury. Superficial fascicles and fibers are more prone to compression than those found more centrally. While large myelinated fibers are more commonly injured than smaller fibers or unmyelinated fibers, no significant difference in the sensitivity of large motor or large sensory fibers to compression has been consistently observed.[101] The propensity of certain fascicles to be injured based on topography and the variability of fascicular arrangements among individuals may lead to mislocalization of injuries when purely clinical assessments are made (Table 9-4). The utility of electrodiagnostic techniques to determine the focus of injury becomes paramount in these situations.

TABLE 9-4

NERVE FASCICLES ESPECIALLY PRONE TO COMPRESSION

Nerve	Region	Susceptible Fascicles
Sciatic	Proximal thigh Pelvis	Peroneal
Common peroneal	Fibular head	Deep peroneal
Ulnar	Elbow	Sensory to medial hand motor to hand intrinsics
Median	Supracondylar	Anterior interosseus
Median	Carpal tunnel	Sensory
Radial	Radial groove	Variable

While the tibial portion of the sciatic nerve is more susceptible to ischemic injury than its peroneal counterpart, compression injury more commonly affects the peroneal trunk. Sciatic injuries following pelvic or proximal femur fractures or hip dislocations may be mistaken clinically for a more distal injury to the common peroneal nerve. Compression injury of the ulnar nerve at the elbow affects fascicles innervating the hand intrinsics and providing sensation to the ulnar aspect of the hand more than it affects fascicles innervating the flexor carpi ulnaris or providing sensation to the dorsum of the hand. A purely clinical assessment may lead to false localization of injury to the wrist. Injury of the common peroneal nerve at the fibular head is more apt to produce symptoms in a deep peroneal distribution. Trojaborg[102] observed variable involvement of the brachioradialis and specific extensor muscles during compression of the radial nerve during sleep or secondary to blunt trauma. Sensory abnormalities were also variable. Selective injury to specific fascicles at the level of the radial groove can only explain the heterogenous patterns because individual branch points occur well below the site of injury. While the anterior interosseus branch of the median nerve takes off 5 to 8 cm distal to the lateral humeral epicondyle, supracondylar fractures commonly selectively injure those fascicles. The posterior location of the fibers, close to the bone at the level of the distal humerus, enhances the likelihood of injury. The variability of sensory symptoms in CTS and the usual late finding of motor weakness suggest a more superficial but arbitrary fascicular topography of median nerve fibers beneath the flexor retinaculum.[103]

Ischemic Injuries

Ischemia is unequivocally a major cause of traumatic nerve injury. Yet, the abundant collateral circulation enveloping peripheral nerves has frustrated attempts by researchers to understand the pathomechanics underlying ischemic nerve injury. Results of many studies have been confounded by the mechanism by which cessation of blood flow was instituted. Most studies have employed tourniquet constriction of limbs. Controversy has been raised as to whether pathologic changes observed in these studies reflect ischemic injury, compression injury, or a combination of the two.[32,104–107] More recently, alternative methods of obtaining a relatively pure ischemic insult have been achieved. Korthals and Wisniewski[108] developed a model employing ligation of the abdominal aorta and femoral artery. This technique was modified by Hess and colleagues[109] and Fowler and Gilliatt[110] in several studies. Eloquent studies[111] utilizing infusions of arachidonic acid have also been developed. Clinically, pure ischemic neuropathy has been a consequence of ergot toxicity.[112]

Pathophysiology

The pattern and character of pathologic change from ischemia differs from that inflicted by other causative agents. Ligation of the internal and external iliac arteries of rabbits led to injuries of the sciatic nerves in the study by Hess et al.[109] The tibial portion of the sciatic nerve was predominantly effective relative to the peroneal portion. A junctional zone extending an average of 20 mm was observed early on and was felt to be the site of ischemic axonal injury. The zone revealed histologic changes that distinguished it from more distal Wallerian degeneration. In addition to axonal and myelin destruction, Schwann cells, fibroblasts, and vesicles were also destroyed.[108] Massing of cytoplasmic organelles was also observed in this region.[113] Just proximal to the zone, a region of paranodal demyelination developed. In 92.5% of the fibers, the last node proximal to the junctional zone sustained paranodal demyelination without evidence of axon injury and extended to the penultimate node in 60.5% of fibers and to the third node in only 22% of fibers. Wallerian degeneration was observed distal to the zone of injury within several days after the insult. In a small percentage of fibers, segmental demyelination occurred without evidence of Wallerian degeneration.[109] Segmental demyelination as evidenced by conduction block has also been observed in ischemia caused by ergot toxicity,[112] femoral artery ligation,[114] and arachidonic acid infusion.[115] In contrast to patterns seen in compressive neuropathies, the central core of large fascicles was particularly sensitive to ischemia. These findings suggest that certain regions along the nerve may be more prone to ischemic injury and likely reflect vascular watershed areas.

No consensus has yet developed regarding the tenacity of different types of nerve fibers in the face of ischemia. Lewis et al[116] clinically observed the loss of tactile and position sense early, followed later by the loss of pain and temperature sensation. It was deduced that larger fibers were most susceptible. Yet, it was also observed by several researchers that sensation is always impaired prior to the loss of motor function.[104,106,108,109] Parry and Brown[111] alternatively observed a disproportionate loss of unmyelinated fibers relative to those myelinated. Of interest is that some protection against ischemia has been observed in elderly, diabetics and uremic patients.[107] It is likely that enhanced collateral flow in the face of gradually waning primary vascular channels may help maximize blood flow in these tenuous situations.

Clinical Considerations

While a relatively discrete ischemic neuropathy may be observed in vasculopathies such as polyarteritis nodosum[117] and to some extent diabetes mellitus,[118] it is uncommon to see pure ischemic injury associated with trauma. It has long been suggested, however, that ischemia is the primary cause of peripheral nerve injury associated with compartment syndromes.[105,119,120] A compartment syndrome is defined by Matsen[120] as a condition in which the circulation and function of tissues within a closed space are compromised by increased pressure within that space. Increased compartmental pressure is the fundamental condition underlying many clinical entities, including Volkmann's ischemia, rhabdomyolisis, crush syndromes, and exercise induced ischemia. An initial insult spurs hemorrhage and edema. Compartment pressure rises, collapsing veins and impeding drainage. The arteriovenous gradient is minimized. Decreased blood flow locally leads to ischemia. A vicious cycle of edema, ischemia, and necrosis ensues.

Between 1900 and 1950, several clinicians observed rates of nerve injury associated with compartment syndromes ranging from 50% to as high as 75%.[105] Peripheral nerve tissue is more susceptible to ischemia than other connective tissues.[110] Functional abnormalities commencing in paresthesias and loss of sensation of light touch and pinprick precede motor weakness. Intracompartmental pressures as low as 30 mm Hg have been observed to provoke sensory abnormalities[121] while others have suggested higher pressures in the 40 to 50 mm Hg range.[120] The latitude reflects several variables, including blood pressure, metabolic rate of tissues, and duration of pressure elevation. Sensory abnormalities have been noted within 30 minutes of the onset of ischemia[120] and have been touted as the most sensitive physical findings. They are harbingers of more serious injury if treatment (fasciotomy) is not instituted swiftly.[121] Irreversible changes are observed after 2 to 4 hours of ischemia and have been used as the benchmark for experimental studies.

Electrodiagnosis

Electrodiagnostic findings obtained by Fowler and Gilliatt[110] closely parallel the histologic changes observed by Hess et al.[109] In the majority of cases there was an immediate loss of ability to evoke a motor action potential proximal to the site of injury. Over the course of several days distal stimulation failed to elicit a motor action potential, suggesting Wallerian degeneration. In several animals with less profound functional deficits, distal stimulation was reduced relative to controls, reflecting axon loss; however, no response could be obtained from proximal stimulation. This suggested conduction block in the surviving axons. In general, ischemia produces a mixed injury with axonal loss predominating over demyelination.

While the measurement of intracompartmental pressure is the gold standard, nerve conduction velocity and motor action potentials may provide essential adjunctive information. Matsen and colleagues[119] used themselves as subjects to assess the utility of electrodiagnosis in the monitoring of compartment syndromes. At pressures ranging from 50 to 60 mm Hg, nerve conduction velocity abruptly dropped to 75% of controls at 25 to 30 minutes and paralleled the onset of motor weakness and anesthesia. Amplitudes of motor action potentials also dropped to 75% of distal stimulation in the same time frame. While attempts have been made to establish the minimal pressures at which fasciotomy should be considered, variables including hypotension, impaired vascular flow, concomitant injuries, and increased metabolic demands may place tissues at higher risk of injury at lower than expected compartment pressures. In patients who have sustained multiple trauma and may be hypovolemic or who are obtunded and cannot actively move the limb, electrodiagnostic studies may be helpful. Muscle death occurs earlier than nerve death. However, sensibility and electrodiagnostic correlates of nerve injury in the face of significant compartment pressure occur earlier than muscle injury and are reversible. In other words, nerves are more resilient but their injuries are more readily apparent earlier than muscle injury. Because they reflect the physiologic status of compartmental tissues, they allow for serial, noninvasive monitoring with quantifiable measures and may be the earliest marker of impending tissue injury.

Stretch Injuries

Clinical Considerations

Fractures, dislocations, and fracture-dislocations are commonly complicated by traction injuries to peripheral nerves. While the overall incidence has not been established, the rates of trauma to particular nerves with certain injuries have been assessed. According to Gurdjian and Smathers,[122] upward of 95% of all nerve injuries associated with fractures occur in the upper extremities. Five percent of all shoulder dislocations are complicated by nerve injury (Figure 9-11). In Rowe's series,[123] 30% of injuries were to the ulnar nerve, 18% to the radial nerve, 11% to the axillary nerve, and 4% to the median nerve. Combinations, likely reflecting brachial plexus injury, accounted for 37% of associated nerve injuries and overall are the most common. Mast and colleagues[124] observed an 18% rate of radial nerve palsy associated with humeral shaft fractures (Figure 9-12). Sciatic neuropathy complicates traumatic hip dislocations 11% of the time according to Brav.[125] Peroneal neuropathy occurs in 27% of traumatic knee dislocations (Figure 9-13).[126] The great amount of torque generated by inversion injuries to the

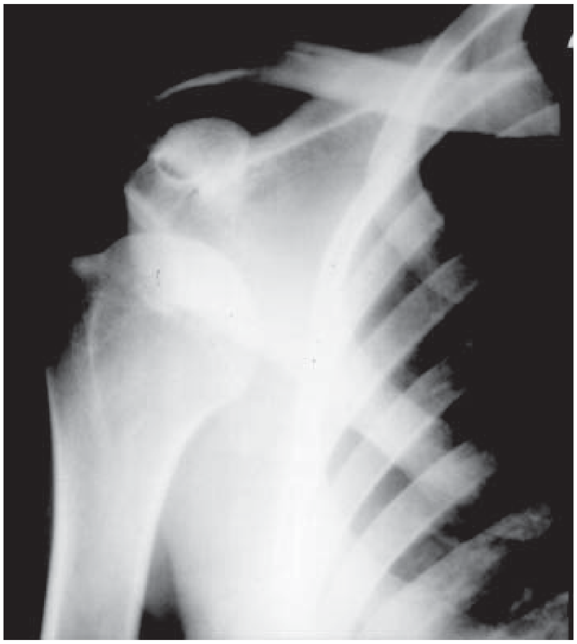


Fig. 9-11. Anterior shoulder dislocation. Stretch injury to major nerve branches or brachial plexus may be associated in 5% of injuries. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

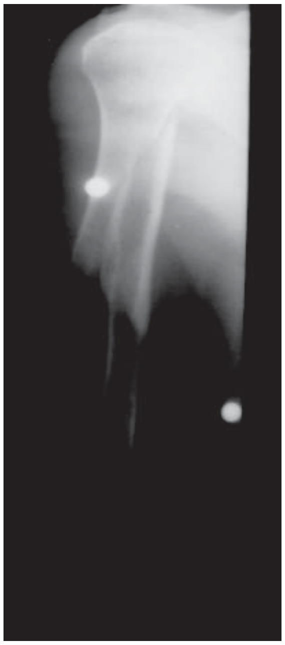


Fig. 9-12. Mid-shaft humeral fracture. Commonly associated with radial nerve injuries. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

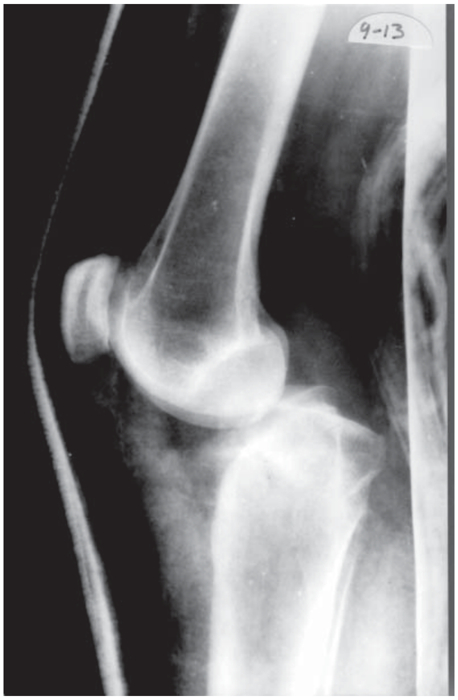


Fig. 9-13. Posterior tibial dislocation following a water skiing accident. Traction injury to the common peroneal nerve or peroneal fascicles of the distal sciatic nerve commonly coexist with this severe injury. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

ankle may lead to proximal fibula fractures and associated peroneal neuropathies (Figure 9-14).[127] Severe varus stress, rupture of the lateral collateral ligament, may also cause a torsion injury of the distal part of the leg resulting in peroneal nerve injury.[128] Stretch is also an element in injuries associated with gunshot wounds[129] and has also been documented[130] as a complication of limb-lengthening procedures utilizing external fixation devices such as those designed by Ilizarov and Wagner.

The distribution of fracture-related nerve injuries has remained remarkably consistent over the years and typifies the intimate relationship of certain nerves and bones. Radial nerve lesions comprise nearly 58% of cases. Injuries to the ulnar nerve constitute 20%, peroneal neuropathies 15%, median neuropathies 5%, and sciatic neuropathies 2%.[122,131,132]

Pathophysiology

The strength and elastic properties of peripheral nerve have been extensively documented.[22,23,25]

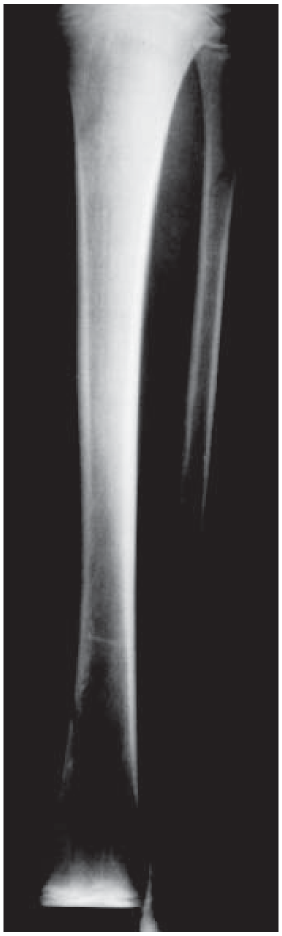


Fig. 9-14. Distal tibial fracture with proximal fibular fracture. May be associated with peroneal neuropathy. Photograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

These variables are important not only in the assessment of potential injury associated with trauma, but also in determining when end-to-end anastomoses should be abandoned in favor of grafting in nerve injury repair. As with other tissues, the strength of peripheral nerves is directly proportional to their cross-sectional area. The cable design of fascicular organization imparts even greater strength. Sunderland and Bradley[26] followed the length of several nerves and observed that the maximum load sustained by the nerve was greatest where the fascicular cross-sectional area was greatest. Total cross-sectional area was held constant. Thus, tensile strength varies not only between different nerves but even between different areas of the same nerve.

The severity of nerve injury associated with traction is related to the extent of elongation, the intensity and duration of the deforming stress. Progressive traction forces placed on a nerve lead to a spectrum of functional decline. Lundborg and Rydevik[133] observed the first evidence of injury when nerves were stretched by approximately 8% of their total length. At this stage, microthrombi and emboli appeared in venules with a diminution of venous flow greater than 50%. A complete halt of arteriolar and capillary flow occurred at 15% elongation. Stagnation of endoneurial lymphatic flow followed, leading to increased endoneurial fluid pressure, decreased oxygenation, and cessation of axonal transport. If continued for greater than 30 minutes, axonal degeneration ensued. Lundborg and Rydevik's[133] study revealed no evidence of endoneurial vessel damage or disruption of the blood-nerve barrier imparted by the perineurium. These findings contradict those of Denny-Brown and Doherty[23] who suggested that high levels of endoneurial fluid were transudates emanating from small damaged blood vessels. They also call to question Haftek's[24] findings of perineurial disruption antedating axonal injury, a claim dismissed by Sunderland.[9] The compromise of vascular flow occurs after the slack of the epineurium, perineurium, and endoneurium have been taken up. As the perineurium stretches, the cross-sectional area of the nerve decreases, not unlike Chinese finger traps. The compression may lead to vascular compromise. While it has not been shown experimentally, it is postulated that direct pressure may also lead to deformation of the myelin sheaths, causing first-degree injury in the mildest of cases. At a maximum of 20% elongation, the elastic limit of the nerve is reached. This is commonly paralleled by physiologic failure. Sunderland[9] suggests that, at this juncture, axonal injury transpires, culminating in second-degree injury and Wallerian degeneration. Further tension disrupts endoneurial connective tissue (ie, third-degree injury) with potential for neuroma formation. As a maximum of 30% elongation is approached, the mechanical limit of nerve integrity is reached and the perineurium fails. Further stretch inevitably ends with epineural destruction and loss of continuity of the nerve trunk.[9]

The pivotal role played by the perineurium in the protection of peripheral nerves against traction forces is further validated when comparison to the nerve roots is made. Nerve roots, which are more prone to traction injury than nerve trunks, possess elastic and mechanical limits 5% less than those manifest in nerve trunks. The lack of perineurial sheathing at the root level is felt to underlie this susceptibility to traction injury.[26]

Prognosis

Outcomes following traction injuries, as would be expected, directly relate to the severity of the injury. Fascicles and individual nerve fibers may sustain widely disparate levels of injury. Integration of fibers of all levels of performance reflects functional outcome. Omer[134] observed nearly 1,000 nerve injuries during his tenure at Brooke Army Hospital, San Antonio, Texas, during the Vietnam conflict from 1966 to 1970. Nerve injury in continuity associated with fractures, fracture-dislocations, or dislocations revealed spontaneous recovery in approximately 85% of cases. Isolated injury above the elbow required on average 2 to 4 months for recovery to ensue. Those below the elbow took 1 to 4 months to recover. Multiple injuries followed similar time frames. While electromyography (EMG) was mentioned in Omer's[134] study, no findings were recorded and thus no objective distinction between neuropraxic and axonotmetic injuries can be made. The fact that 37% of spontaneous recoveries occurred within the first 6 weeks following the injury may be most consistent with a predominant neuropraxia. The rate of recovery is too rapid to be explained by reinnervation. The remaining 63% required 10 to 32 weeks to fully recover. They presumably experienced axonotmetic or second-degree lesions by Sunderland's[34] criteria. These findings are consistent with those of Seddon[31] who also found that nerve injuries associated with fractures were predominantly axonotmetic.

Cold Injuries and Exposure

The ravages of cold-induced injury have been well documented during military campaigns of the last 2 centuries. Barron Larrey, chief surgeon of Napoleon's Grande Armée, documented the effects on French troops during their ill-fated assault on Russia.[135] The 115,000 cases of "trenchfoot" suffered by British soldiers wallowing in the cold, dank mire of the trenches of World War I significantly hampered the army's military effectiveness.[135] Ungley and Blackwood[136] astutely documented the clinical features and pathophysiology of the distal vasoneuropathy termed *immersion foot,* which was sustained by myriad troops exposed to cold sea water for extended periods during World War II. Immersion foot and trenchfoot share the same clinical and pathological presentations and are felt to be the same syndrome. Despite an enhanced insight into cold-induced nerve injuries, they continue to pose a significant threat to both military and civilian populations. Cold injuries accounted for nearly 20% of all British casualties sustained during the Falkland war with Argentina in the early 1980s.[137] More recently, immersion foot has become a problem manifest in the homeless population in the United States.[138] Iatrogenically induced cold injuries have also been documented during cryotherapy for musculoskeletal injuries.[139]

Severe exposure to cold commonly results in freezing of the tissue with subsequent necrosis. Denny-Brown and colleagues[140] observed that direct freezing of rabbit sciatic nerve for 5 minutes led to complete necrosis of all involved fibers. Endoneurial structures were destroyed with the exception of the endothelial lining of penetrating blood vessels. When direct freezing was limited to under 2 minutes, selective involvement of fibers was observed. Large myelinated motor fibers were more susceptible to injury than myelinated sensory fibers and both were more susceptible than unmyelinated fibers.

The absence of neuroma formation or scarring at the site of freezing suggests that relatively good recovery may be anticipated. Denny-Brown et al[140] observed evidence of regeneration within 1 to 2 months and completion by 3 months.

Clinical Considerations

Less intense cooling to temperatures just above freezing leads to a unique syndrome termed *trench foot* in World War I, *immersion foot* in World War II. In the Ungley and Blackwood series,[136] men exposed to immersion of their limbs in sea water at temperatures ranging from 1°C to 9°C for upward of 14 days manifest the now classic syndrome-immersion foot. Three stages have been described (Table 9-5).

Stage I prehyperemic. In the prehyperemic stage, the extremities are cold, swollen, and numb. Anesthesia in a stocking or glove distribution is dense. Distal pulses may or may not be present. Stage I commonly lasts from hours to days.

Stage II hyperemic. Stage I is followed by a hyperemic stage, which may last 6 to 10 weeks. Several findings dominate the clinical picture. Presumed injury to autonomic nerve fibers leads to vasomotor instability. The temperature of the affected limbs remains elevated from 30°C to 34°C, with loss of the distal-proximal temperature gradient. The limb remains erythematous with a violaceous tint. The color change is enhanced by dependency and blanches with elevation. Swelling is observed, especially in limbs that have been inadvertently warmed rapidly.

Sensory abnormalities often reach their nadir at 7 to 10 days after rescue and remain sustained at this intensity for more than 6 weeks. Parasthesias

TABLE 9-5

CLINICAL PRESENTATION OF NONFREEZING COLD INJURY

Stage	Duration	Presentation
Stage I Prehyperemic	Onset to several hours or days	Stocking or glove anesthesia Edema, Hypothermia ± Pulselessness
Stage II Hyperemic	End of stage I to 6–10 weeks	Temperature elevation Erythema, Edema Pain: Dysesthesias, Allodynia, Hyperpathia, Lancinating Pain
Stage III Posthyperemic	End of stage II to years	Normalization of temperature control Persistent hypersensitivity to cold Raynaud's phenomenon Hyperhidrosis Improving sensory and motor function

are universal in a stocking or glove distribution and are intensified by dependent positioning of the limb. Shooting and stabbing pains have also been noted. They are more intense with dependency and may be exacerbated by exposure to cold or warmth, exercise, micturition, defecation, or yawning. Commonly, pain complaints are increased at night. Hyperesthesia and allodynia are also commonly experienced. Sensitivity to pinprick, light touch, and vibration are variably decreased or lost.

Motor strength of muscles within the cooled territory is commonly impaired. Anhydrosis coincides with the extent and distribution of sensory loss. Blistering may be observed in moderate to severe cases as early as 3 days postrescue but are more commonly seen around the 8th to 10th day. Healing may be protracted and take from weeks to several months. Hair, fingernails, or toenails may also be lost.

Stage III posthyperemic. During stage III, which can last for weeks to years, vasomotor control begins to normalize. Skin temperature and color return to normal. There remains, however, a sensitivity to cold that may last for months to years. This abnormal response to cold and emotional stressors is termed Raynaud's phenomenon, and is characterized by vasospasm causing painful blanching, followed by cyanosis. As the vasospasm remits, the digits become erythematous.[141] Hyperhidrosis may develop irrespective of environmental temperature. Sensory and motor functions continue to improve.

Prognosis

Prognosis following immersion foot is generally good. Mild cases resolve within 2 to 5 weeks. In fact, the vast majority of cases in White's[142] series returned to duty within 6 weeks. In those more severely injured, a more protracted convalescence may take place lasting 3 to 12 months. Of the 18 patients followed long-term in the Ungley and Blackwood[136] series with moderately severe courses, 35% returned to full duty. Fifty-four percent returned to light duty, limited by cold sensitivity and standing tolerance. Only 11% were unable to return to some form of active duty. Long-term sequelae included intermittent shooting pains in 64%, paresthesia with prolonged standing in 35%, and intermittent swelling in 43%. Sensitivity to cold remained in the majority up to 2 years postrescue.

Pathophysiology

As with other causative agents, the severity and completeness of cold-induced injuries vary with the duration and intensity of exposure. Susceptibility to injury also differs with nerve morphology. Discrepancies in the literature abound and may reflect the methods by which investigators sought to study these questions. Many investigations based conclusions on clinical findings (eg, loss of movement or response to sensory stimulation) while others have focused on the ability to evoke

SNAPs and compound motor action potentials (CMAPs). Further confusing the picture are conclusions based on findings obtained very soon after the cooling event.

Temperature

The study by Schaumburg and colleagues[143] of direct cryoprobe cooling of cat sciatic nerve evaluated decrementing temperature exposure from 20°C down to 6°C. Motor weakness was observed at 10°C. Little structural damage was noted until 7°C was reached. Response to light touch was lost prior to loss of pinprick, which waned at 7°C. Nerve conduction velocities were measured and noted to be delayed at 7°C. The studies were obtained with needle stimulation and recording electrodes and therefore no assessment of action potential amplitudes can be made. Denny-Brown and colleagues[140] also showed substantial injury to rabbit sciatic nerves when directly exposed to temperatures ranging from 5°C to 8°C. Similar findings were reproduced by other researchers.[144,145] Recently, Kennett and Gilliatt[146] noted a significant difference between rabbit tibial nerves directly cooled at 5°C and 1°C for 2 hours. The amplitude of the compound motor unit action potential (CMUAP) of the former decreased by 50% from baseline by the third postcooling day. The amplitude of the latter dropped by over 70%.

Duration

The duration of nonfreezing cold exposure necessary to effect peripheral nerve injury understandably hinges on whether cold is applied directly or through the insulation of skin, subcutaneous fat, and muscle. Direct cooling of rat sciatic nerve to 3°C for as brief a period as 5 minutes results in an inability to propagate an impulse from a proximal point of stimulation. In the study by Nukada and colleagues,[145] normal latencies returned within 30 to 40 minutes; however, a persistent amplitude drop of greater than 40% remained, suggesting axonal injury. Denny-Brown and colleagues[140] observed a spectrum of injury to fibers exposed directly to temperatures 5°C to 8°C for 30 to 120 minutes. Schaumburg and colleagues[143] observed histologic changes when nerves were directly exposed at 10°C for at least 1 hour but not at 30 minutes. At cooler temperatures, even those exposed for only 30 minutes developed substantial injury. More recent studies[144–147] have used 2 hours as a standard duration to provoke direct nerve injury.

In the more clinically oriented studies during the two World Wars, cold exposure for as short a period as 14 hours was sufficient to cause nerve injury. Most soldiers, however, were exposed for several days.[136,142] This was confirmed experimentally by Kennett and Gilliatt.[146] Rabbit hind limbs immersed in 1°C solution for less than 9 hours developed no appreciable injury. Variable pathology was observed between 9 and 14 hours. Injury similar to that seen with direct cooling at 2 hours was observed when the limb was cooled in excess of 14 hours.[147]

Type of Nerve

As with the other variables discussed, discrepancies in the literature regarding differential sensitivity to cold injuries between different nerve types are widespread and likely reflect different methods used in some of the earlier studies. Denny-Brown and colleagues[140] and Schaumburg and colleagues[143] monitored the loss of motor and sensory function. In both studies, the investigators observed paralysis of muscles innervated by the involved nerves at temperatures below 7°C and 10°C, respectively. Sensory loss occurred later at 0°C and 5°C, respectively. It was inferred that large myelinated fibers were more susceptible to cold than smaller myelinated fibers and both more easily injured than unmyelinated fibers. Recent studies[143,146] employing electrodiagnostic techniques as well as electron microscopy substantiate the clinical findings of Denny-Brown and colleagues.

Morphologic Changes

Direct exposure to nonfreezing cold induces characteristic morphological changes in the peripheral nerve. Axonal swelling with clumping of electron dense material can be seen by electron microscopy as early as 6 hours after exposure. By 24 hours, profound subperineurial and endoneurial perivascular edema is present. Disruption of the myelin sheath into ovoid masses and an invasion by macrophages with associated degeneration of the distal axon commences between the third and seventh day. While patchy demyelination was seen in several axons in a number of studies, the vast consensus is that demyelination is not a primary pathologic process.

Electrodiagnosis

Electrodiagnostic studies performed by Kennett and Gilliatt[146] corroborate the finding histologically of Wallerian degeneration as the principal pathology. One hour following cooling, motor action potentials could be evoked below the level of the injury. No potential could be obtained during stimulation proximal to the lesion. Prior experiments[146] that did not serially perform nerve stimulations for periods beyond the acute injury may have misconstrued this as conduction block or focal demyelination. Recordings on the second and third postinjury days revealed a progressive loss of motor action potential amplitude. By the third day, distal amplitudes resembled those evoked proximally. Follow-up studies[146] revealed improvement of amplitude both proximally and distally from almost 45% of preinjury at day 3 to nearly 75% of preinjury at 4 weeks. It was felt that the improvement reflected collateral sprouting as not enough time had elapsed for reinnervation from regenerating fibers to occur.

Generalized cooling by immersion revealed intriguing results. The main sites of injury were not at the distal ends of the nerve fibers but at well demarcated areas at the junction of the middle and upper thirds of the tibia, correlating to the area just below surface level. It was postulated that the abrupt temperature gradient may be the affecting variable.[147]

While the type of injury induced by nonfreezing cold has become discernible, the means by which this damage occurs remains obscure. It has been suggested by Bausbaum[144] that several mechanisms may come into play. Low temperatures may increase the viscosity of lipids within the axon membrane and alter its selective permeability properties and ability to propagate impulses. Another mechanism, which has been well described, is the suspension of axonal transport associated with cooling. Nukada and colleagues[145] showed that by 3 to 5 days after cooling, the blood-nerve barrier had been breached. The increased permeability coupled with poor endoneurial lymphatic drainage leads to vasogenic edema, which may also compromise axonal transport and other metabolically driven mechanisms. An inability to clear accumulating waste and toxins may also play a role.

Penetrating Injuries

The passage of high- and low-velocity missiles through soft tissue produces a wide array of devastating injuries. Almost 70% of penetrating wounds evaluated by the Physical Medicine and Rehabilitation Service at Walter Reed Army Medical Center during the Persian Gulf War were associated with peripheral nerve injuries.[3] Injuries caused by projectiles are undoubtedly the most common of all war-related trauma. A complete discussion of this topic is beyond the scope of this chapter but can be found in the Textbook of Military Medicine volume dedicated to injuries associated with conventional warfare, *Conventional Warfare: Ballistic, Blast, and Burn Injuries.*[148]

Pathophysiology

Elegant models developed by Harvey et al[149] at Princeton University during the 1940s simulated the primary and secondary destruction caused by the passage of high-velocity missiles. As the projectile enters the body it pulverizes all soft tissues in its path, transforming them into a bloody pulp. Immediately following the projectile, a large, subatmospheric cavity develops, whose volume is many times that of the primary missile track. The cavity rapidly expands and contracts, producing severe stresses and strains in the adjacent soft tissues. Secondary damage to the soft tissues surrounding the cavity occurs because of the initial rapid stretch and subsequent recoil.[149] Oscillating shock waves, moving at the speed of sound have also been measured in regions of the body distant from the site of impact.[150]

Peripheral nerve injury caused by penetrating wounds can be attributed to three different mechanisms. Large segments of nerves may be destroyed by direct contact with the missile. Commonly, a complete, fifth degree injury occurs in these situations with expansive gaps between nerve endings observed on inspection. Damage can also occur without a direct hit to the nerve. Nerves, like other components of soft tissue, are markedly displaced and deformed by the large cavity following missile passage. A spectrum of stretch and compression injuries ensues. Puckett et al[151] observed effects on the sciatic nerves of cats subjected to the impact of high-velocity spheres whose impact velocity ranged between 3,200 and 4,100 ft/sec. The rate of soft tissue displacement by the temporary cavity was one tenth of the impact velocity (300 to 400 ft/sec). In 4 of 22 cases in which the sciatic nerve was not hit but was within the zone of soft tissue pressure wave expansion, the nerves remained grossly in continuity. The rapid displacement of the nerve led to a wide variety of histological changes. Destruction of axon cylinders with maintenance of surrounding connective tissue (ie, second degree injury) was the mildest form of injury observed. Commonly, regions of intrafascicular and interfascicular fiber destruction was noted (ie, third and fourth degree injuries). Nerve conduction studies (NCSs) performed within several hours of impact revealed an inability to propagate an impulse from the level of the wound or slightly proximal to it. Puckett et al[151] also observed 15 subjects in which the ability to propagate an impulse across the injured region was maintained. It was presumed that no injury occurred in these nerves because they were outside the perimeter of the temporary cavity. The extent to which cavitation causes injury to peripheral nerves remains unclear. Unfortunately, no microscopic evaluations or changes in the morphology of the evoked potentials were mentioned. Thus, it cannot be deduced as to whether milder (eg, first degree) or incomplete injuries to these nerves were truly experienced. An alternate explanation suggests that electromechanical effects caused by cavitation may cause the neurodysfunction.[148]

The prospect of neurological injury in parts of the body well distant from the site of impact remains controversial. Initially raised by Harvey et al[152] and later confirmed by Berlin[153] and Tikka et al,[154] current consensus in the field remains polarized with as many supporting the theory as opposing it. Suneson et al[155] recorded pressure waves of injuriously high energy and amplitude, moving at the speed of sound not only in the abdomen and thorax of pigs wounded in one thigh but also in the brain and contralateral thigh. While macroscopic integrity of the contralateral sciatic nerve was maintained, electron microscopy revealed distortion of myelin sheaths as well as axons. Microtubules were unusually sensitive, experiencing disarray and disintegration.

Clinical Considerations

Prognosis following penetrating injuries depends not only on the integrity of the peripheral nerve but also on the extent of concomitant injury to muscles, bone, and vascular structures. While data obtained in the civilian sector regarding shotgun injuries cannot be directly generalized to reflect the military experience, some important parallels can be made. Fifty-nine percent of shotgun injuries to the upper extremities included nerve damage.[156] Nerve injury occurred at a rate of 40% when all gunshot wounds were included and 50% when all penetrating trauma was assessed.[157] Of the shotgun wound cases, 60% required skin or pedicle grafts to cover soft tissue defects, fractures occurred in 64%, and vascular disruption was observed in 33%.

Other studies[158,159] measuring the association of nerve injury and vascular injury have an observed rate of 37% in close-range shotgun injuries and 51%[158] for all penetrating trauma. According to Visser and colleagues,[157] data from the Vietnam vascular registry documented a 44% incidence of concurrent nerve and vascular injury. When broken down by extremities, both civilian and military sectors convey a preponderance of complex injuries involving the upper extremities. Over 70% of upper extremity vascular injuries from projectiles were complicated by peripheral nerve injuries. Lower extremities sustaining vascular injuries manifest nerve damage in 32% of civilian cases and 25% of military casualties.

Omer[134] assessed outcomes of nerve injuries from high- and low-velocity gunshot wounds sustained during the Vietnam War. Good outcomes were defined as return of sensibility to pressure of less than 3.84 (0.693 F/gm) by Semmes-Weinstein pressure esthesiometry and manual muscle-testing grade of fair or better. Omer's findings were consistent with those obtained by Foerster in World War I and by Sunderland in World War II (as cited in Omer[129]). In all three conflicts, nearly 70% of nerve injuries associated with gunshot wounds recovered spontaneously to a good level.

Isolated low-velocity (1,000–1,500 ft/s at impact) nerve injuries above the elbow required on average 4 to 7 months to recover, while those below the elbow recovered more rapidly, in 3 to 6 months. When more than one nerve was involved above the elbow, recovery averaged 5 to 8 months. Multinerve injuries below the elbow averaged 3 to 7 months to recovery. A bimodal distribution is observed when all low-velocity injuries are included. A majority recover within the first 6 weeks and a second peak is noted at the 5 to 6 month mark. These differences may reflect primarily neuropraxic injuries in the former and axonotmetic lesions in the latter.

High-velocity (2,500–3,000 ft/s at impact) nerve injuries differed little in recovery time in relation to location above or below the elbow. Isolated injuries required 3 to 6 months for recovery and multiple injuries averaged 5 to 9 months.

These findings are more optimistic than those conveyed in the civilian shotgun literature in which transection, nonfunctional, and no recovery groups comprised nearly 80% of outcomes.[156] Eighty-six percent of vascular injuries complicated by peripheral nerve injuries in civilian cases culminated in significant functional impairment.[157]

The differences in outcome may likely be explained by ballistics. Shotgun injuries are of lower velocity, on average 1,200 ft/s. The blast and shock wave effects are less profound than those generated by high-velocity missiles. The local effect is more injurious, however, as upwards of 275 14-mg shots are concentrated into a small area. As the shots encounter different tissue planes and densities, their trajectories may change. A wide area of local trauma occurs. While outcome data are not yet available, injuries caused by modern ordinance, which spray small fragments at low-velocities, may be more similar to those experienced in shotgun injuries than to the high-velocity missile injuries of previous wars.

ELECTRODIAGNOSIS OF PERIPHERAL NERVE INJURIES

General Principles

Electrodiagnosis is a special form of diagnostic test designed to assess the integrity of peripheral nerves, neuromuscular junctions, or muscles. It is an extension of the history and physical examination and is not intended to be performed in isolation. It is, however, intended to augment the information obtained through interview and careful examination of the patient. Electrodiagnostic testing offers qualitative and quantitative information regarding severity and distribution of nerve injury or disease and information that may be helpful in determining prognosis. The quality and accuracy of an electrodiagnostic study is highly dependent on the expertise and experience of the electromyographer.

It is imperative that meticulous attention to proper technique be employed at all times to ensure accuracy and reproducibility. Although guidelines for performing a specific type and number of tests for a suspected clinical problem have been established by the American Association of Electrodiagnostic Medicine (AAEM), each electrodiagnostic evaluation must be tailored to the patient's presentation. It is not uncommon to alter the approach to a clinical problem during the course of the test as new information is gathered and a new differential diagnosis is entertained.

Electrodiagnosis offers a physiologic perspective; imaging studies, such as magnetic resonance imaging or computed tomography scans, offer an anatomic view of suspected pathology. It is important to recognize and document electrical abnormalities in nerves or muscles. It is particularly critical to understand the clinical implications of these electrical abnormalities in order to properly interpret the findings. General categories of pathology can be detected by an appropriately designed electrodiagnostic study; classes of disorders detectable by electrodiagnosis include the following:

- disorders of the motor neuron,
- diseases of the roots and plexus polyneuropathies,
- entrapment and mononeuropathies,
- neuromuscular transmission disorders, and
- myopathies.

Electrodiagnosis is separated into two general types of electrical studies: NCS or EMG. The term EMG is sometimes used to refer to both nerve conduction studies and electromyography. These studies provide the foundation on which more sophisticated studies such as repetitive nerve stimulation, single fiber, macro EMG, and somatosensory studies are performed.

Neurophysiology

Motor Unit

The motor unit is defined as an anterior horn cell, its axon and corresponding terminal branches, the associated neuromuscular junctions, and all muscles fibers innervated by the axon. It is the small functional element of contraction.[159] Discharge of a motor unit results in synchronous contraction of all muscle fibers innervated by the axon. Pathology may occur at any point or segment along this path and is potentially detectable with selective electrodiagnostic studies.

The average size of a motor unit can be expressed as the ratio between the total number of extrafusal fibers and the number of innervating motor axons. This is referred to as the innervation ratio:

$$\text{Innervation ratio} = \frac{\text{Total number of muscle fibers}}{\text{Total number of innervating motor axons}}$$

Motor axons vary in the number of muscle fibers they innervate depending on the type of muscle involved. For example, extraocular muscles have few muscle fibers per axon (ratio of 3:1), while the gastrocnemius muscle has hundreds of muscle fibers per axon. Those muscles with a small ratio are involved in fine gradations of movement, while those with larger ratios produce relatively coarse movements.[160] Motor units also vary in their size. Some have thick and extensively myelinated axons while some are unmyelinated. The heavily myelinated axons conduct faster than the unmyelinated axons. Large diameter, myelinated axons conduct impulses at about 60 m/s, while small diameter axons with less myelin conduct at about 45 m/s.[161]

The larger motor units have a higher innervation ratio, greater twitch tensions, faster twitch contractions, and a greater tendency to fatigue.[162] The size principle of Henneman[163] proposes that motor units are recruited in an orderly fashion with small motor neurons activated first, followed by progressively larger motor neurons. Thus, mild contraction efforts reflect activation of smaller motor units while progressively increasing contraction force is the result of activation of larger motor units. With few exceptions, the Henneman size principle is applicable to virtually any voluntary activation of motor units.[164,165]

Motor neuron disorders and neuropathy, for example, typically do not alter the size principle. However, an exception to the rule may include the random recruitment pattern generated after reinnervation has occurred in a previously transected peripheral nerve.[166] This loss of the size principle response is attributable to the misdirection of motor axons and the associated impairment or loss of orderly recruitment. It is possible to reestablish the size principle after transection of a nerve if the affected motor axons can reinnervate their respective original muscle fibers.[167] Histologically, all muscle fibers of a given motor unit share the same characteristics. The muscle fiber territories of different motor units overlap so that muscle fibers of a single motor unit are in contact infrequently with fibers innervated by the same motor unit.[160]

Membrane Potential

Muscle membranes polarize at –90 mV while nerve axons polarize at –70 mV.[168] Suprathreshold stimulation results in generation of an action potential due to increased sodium conductance. An all-or-none phenomenon occurs after threshold depolarization, independent of the type of stimulus applied.

An electrical stimulator has a cathode, or negative pole, and an anode, the positive pole. Electrical stimulation generates negative charges under the cathode external to the axon membrane, causing a relative increase in the positive charges internal to the axon membrane at the same point along the axon. This process is referred to as cathodal depolarization. Under the anode, negative charges leave the membrane surface, resulting in greater negativity inside the cell and creating anodal hyperpolarization. With depolarizations of 10-30 mV, threshold of the membrane potential is reached and an all-or-none action potential is generated. The action potential propagates in both directions along the nerve from the point of stimulation.

Strength–Duration Relationship

The shorter the duration of electrical stimulation, the greater the intensity required to produce the same degree of depolarization. The strength-duration curve illustrates this principle graphically. Rheobase is the minimal current strength below which no response occurs even if the current lasts greater than or equal to 300 ms. Chronaxie is the minimum duration of current necessary to excite the cell at twice the rheobase. The use of chronaxie and rheobase to determine nerve excitability is somewhat anachronistic as more precise and useful techniques have been developed over the past several decades.[160]

Motor neurons have an inherent excitability that correlates with the axon diameter, conduction velocity, and motor unit size. An action potential can be propagated at greater speed if the four following conditions are present[160]:

1. faster rate of action potential generation,
2. increased current flow along the axon,
3. lower depolarization threshold, and
4. higher temperature.

Myelinated fibers propagate action potentials via saltatory conduction, which involves current "jump" from one node of Ranvier to another. This is in contrast to the continuous propagation of impulses along unmyelinated fibers. The greater the myelin thickness, the less the internodal capacitance and conductance. As a result, for a given axon diameter, the greater the myelin thickness, the faster the conduction velocity.

In demyelinating conditions, internodal capacitance and conductance increase, inhibiting action potential propagation. If sufficient inhibition occurs to cause failure to activate the next node, conduction block occurs. If inhibition is incomplete, conduction velocity may be slowed with temporal dis-

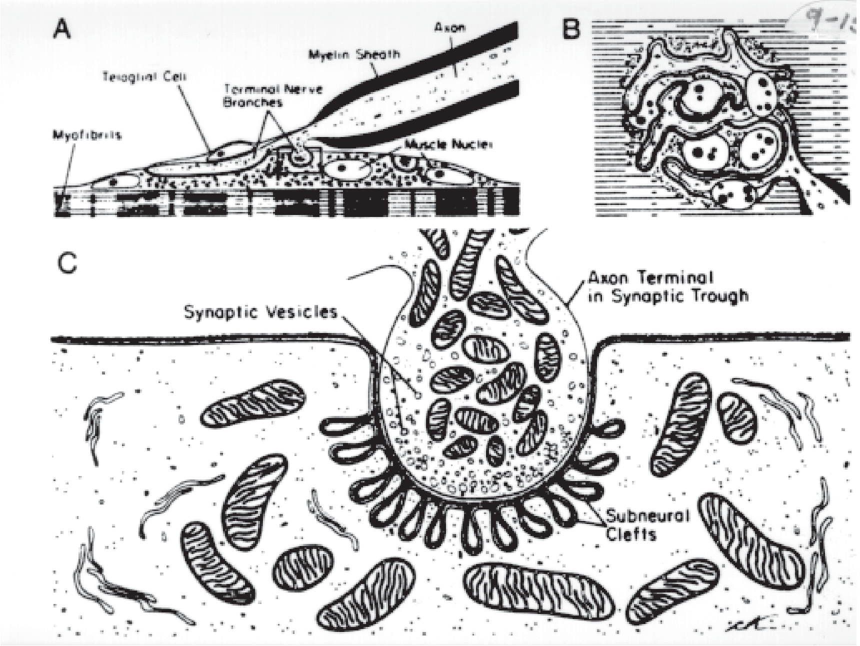


Fig. 9-15. Schematic illustrations of the neuromuscular junction. **A, B**: Light microscopic views of the motor endplate. **C**: Cross-sectional electron microscopic view of the motor endplate. Neurotransmitter is released from the neural synaptic vessels into the cleft. Interactions between neurotransmitter quanta and postsynaptic receptors result in subthreshold depolarization, termed miniature endplate potentials. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 171.

persion of the action potential. In focal demyelinating conditions due to compression, the capacitance of the internodal membrane may actually be decreased due to the pathological narrowing of the fiber diameter. As an isolated change, this would facilitate impulse conduction. However, increased resistance to action potential propagation secondary to the compression greatly outweighs this effect and results in slowing or block of conduction from one node to the next at the site of compression.

An impulse is conducted electrically from the anterior horn cell to the neuromuscular junction. At the junction, transmission is continued via the presynaptic release of acetylcholine (Figure 9-15). The acetylcholine then binds with acetylcholine receptors on the postsynaptic membrane, generating an electrical impulse. If a sufficient number of acetylcholine molecules are released and successfully bind with the receptors, the impulse then propagates down the muscle fiber at 4 to 7 m/s.[161,169]

Instrumentation

NCSs and EMG are performed on highly sophisticated instruments. Many of these instruments are equipped with computers for rapid acquisition and display of electrodiagnostic data. Some are capable of storing and retrieving both numerical information and graphs. The electrodiagnostic machine is composed of multiple integrated components (Figure 9-16).

At the very minimum, the instrument should be able to display latency and amplitude of a SNAP or CMAP. The duration of the action potential may be

Fig. 9-16. Modern computer based electrodiagnostic machine. Includes preamplifier, differential digital amplifier, stimulating electrode, color monitor, audio speakers, printer, and floppy disk memory storage.

calculated or automatically recorded. The measurement of the area under the curve of the action potential may be desirable and is available on some electrodiagnostic instruments. All such devices have a range of frequencies over which electrical activity is recorded. Some offer only selected ranges, while others permit manual selection of a broad range of low to high frequencies depending upon the desired result.

Electrodiagnostic studies involve the recording, amplification, filtering, processing, display, measurement, and interpretation of biologic electrical data detected by surface or needle electrodes. The data obtained through EMG involve needle electrode pickup of volitional motor unit action potentials and spontaneous potentials arising from muscle membrane instability. Nerve conduction studies involve electrical stimulation of sensory or motor fibers or both, and pickup of time-locked sensory, motor, or mixed action potentials at a separate, accessible point along the nerve or muscle.

Action potentials are generated in the human body, which acts as a volume conductor. Electrical signals detected by the electrodes are first amplified and filtered to eliminate environmental distortion of the desired action potentials. Real time or analog signals are transformed into digital data displayed as waveforms on the monitor of an electrodiagnostic machine. The visually displayed data are accompanied by acoustic data corresponding to the analog signals received.

Waveform display of an action potential, by definition, requires two electrodes because an action potential is defined as the difference between two electrodes locations in a volume conductor. Action potential amplitudes are expressed in millivolts and microvolts; currents are expressed in milliamperes and microamperes; and impedances in kilohms and megohms. Latency and duration of action potentials are measured in milliseconds and microseconds.

Compound motor action potentials are obtained by placing an active electrode on a muscle's motor point. The reference electrode is placed on a relatively electrically inactive site, such as the tendon of the muscle, in order to maximize the waveform amplitude. If the active and reference electrodes are placed in similarly electrically active sites, then little differential amplification occurs, and the resulting waveform amplitude will be significantly attenuated.

Sensory nerve action potential waveforms appear to be optimally displayed when the distance between the active and reference electrodes is greater than the spatial extent of the potential's rise time. Thus, an interelectrode separation of greater than or equal to 4 cm is preferred in order to obtain maximum waveform amplitude. An interelectrode distance less than 4 cm results in reduction of the action potential due to greater common mode rejection.[170,171]

Amplification

Amplification is expressed as a gain or sensitivity factor. The gain is the ratio of the amplifier output to its input signal. An input of 10μV, which results in an output of 1 V, has a gain of 100,000.[171,172] Sensitivity is a ratio of input voltage to the size of deflection of the cathode ray tube (CRT).[171] It is recorded as the number of microvolts or millivolts per division. A division on the CRT grid is typically 1 cm. Electrodes and amplifiers form a circuit dependent on Ohm's Law, which states that voltage (E) is equal to the current (I), multiplied by the resistance (R). Thus:

$$E = I \bullet R$$

Impedance (Z) is substituted for resistance (R) when dealing with alternating currents. Ohm's Law is modified in the following fashion:

$$E = I \bullet Z$$

The electrode and amplifier form a series circuit in which both have an impedance value. Because each part of the circuit has an impedance, the total voltage drop across such a circuit is due to the summation of voltage reduction across each circuit component. The voltage of the observed signal is directly proportional to the impedance of the amplifier and indirectly proportional to the sum of the impedance of the electrode and amplifier.[172] The higher the impedance of the input amplifier, the less the impact of the electrode impedance. Thus, a signal is maximized if the impedance of the amplifier (typically up to hundreds of megohms) is substantially greater than the impedance of the electrode (typically recorded in kilohms).

Differential Amplification

Differential amplifiers are used in electrodiagnostic machines for the purpose of amplifying the difference between input from active and reference electrodes (difference mode signals), while canceling similar signals (common mode signals). The differential amplifier requires an input terminal for an active and reference recording electrode, as well as an input terminal for a ground electrode. Although no two electrodes have exactly equal impedance, it is important to minimize the electrode impedance difference in order to optimally reduce common interference signals. Also, a high amplifier input impedance serves to minimize the effect of electrode differences in impedance.

The common mode rejection ratio (CMRR) is the ratio of the output of the amplifiers when a signal is amplified differentially vs that present in the common mode.[172] It provides a means of identifying the differential amplification between the signal and the common mode voltage.[160] Most electrodiagnostic equipment has a CMRR greater than or equal to 10,000. According to Kimura,[160] differential amplifiers should have a CMRR exceeding 100,000. However, he points out that even high CMRRs may not completely eliminate extrinsic interference for the following two main reasons:

1. Although electromagnetic interference similarly affects each electrode, there is a variable degree of difference depending on the position of the electrodes.
2. There is also a variable degree of difference between the electrodes depending on their unequal electrical contacts.

Use of short, well-shielded electrode cables and proper grounding of the patient, bed, and electrodiagnostic instrument are important measures to reduce electrogmagnetic interference.

Filters

Action potentials generated in electrodiagnostic studies are the result of a summation of sine waves of variable amplitudes and frequencies.[170,171] High frequencies are manifested by the rapidly changing components of a waveform, while low frequencies are found in the slowly changing portions of a waveform. In waveforms obtained with NCSs, the rapidly changing portions of the waveforms include baseline take-offs, inflection points, rise time, and peaks. Slowly changing components include baseline returns and total potential durations. In EMG, the initial and terminal segments of the CMUAPs and the terminal portions of positive sharp waves constitute low-frequency components.[172]

Filters permit the recording of the desired signal by allowing input of all frequencies of interest contributing to the intended signal, while eliminating as much electrical interference as possible. Thus, this process helps to minimize contamination of the desired signal by extraneous electrical input. A high-frequency (low-pass) filter shunts high frequencies, but passes low frequencies. A low-frequency (high-pass) filter blocks low frequencies, but allows passage of high frequencies.

Different low- and high-frequency filter settings are required for different forms of electrodiagnostic evaluation in order to maximize recording of desired frequencies. The low-frequency value indicates the lowest frequency to which an amplifier will respond, while the high-frequency value indicates the highest frequency detectable by the amplifier.[171] Recommended filter settings include the following: 2 to 10 Hz low frequency to 10,000 Hz high frequency for motor NCSs; 2 to 10 Hz low frequency to 2,000 Hz high frequency for sensory NCSs; 20 to 30 Hz low frequency to 10,000 Hz high frequency for routine EMG; and 500 to 1,000 Hz low frequency to 10,000 to 20,000 Hz high frequency for single fiber EMG. Somatosensory evoked potential filter settings are typically 1–10 Hz low frequency to 500 to 3,000 Hz high frequency (Table 9-6).[172]

Changes in the low- and high-frequency filter settings result in alteration of the sensory and motor action potentials obtained. For instance, progressively increasing the low-frequency filter when evoking a SNAP results in a progressive decrease in its amplitude, peak latency, and negative spike duration, but possible increase in its overall duration, particularly if an extra phase occurs in the terminal portion of the signal. If the low-frequency filter is set too low, it results in an unstable baseline.

TABLE 9-6

COMMON FILTER SETTINGS FOR ELECTRODIAGNOSTIC STUDIES

Test	Low Frequency (Hz)	High Frequency (Hz)
Motor nerve conduction	2 to 10	10,000
Sensory nerve conduction	2 to 10	2,000
Standard EMG	20 to 30	10,000
Single fiber EMG	500 to 1,000	10,000 to 20,000
Somatosensory evoked potentials	1 to 10	500 to 3,000

EMG: electromyography

If the high frequency filter is set too low, the amplitude of the high-frequency or rapidly changing components of the evoked action potential is reduced.[160,171,172]

Frequencies obtained by needle electrode (electromyography [EMG]) may range from 2 Hz to 10,000 Hz, although as noted previously, some prefer 20 to 30 Hz to 10,000 Hz. Increasing the low-frequency cutoff value from 2 Hz to 32 Hz suppresses slowing changing components of a CMUAP. Fibrillations and insertional activity contain primarily high-frequency components and are, thus, not significantly affected by raising the low-frequency cutoff value. However, the tail of the positive sharp wave, a slow-moving component of this potential, will be distorted with progressive increase in the low-frequency component.

Electrodes

Electrodiagnostic evaluations require three electrodes, one active, one reference, and one ground electrode. Surface electrodes placed over muscle motor points in NCSs record summated electrical activity from many motor units. Needle electrodes in EMG pick up individual motor unit action potentials generated within a restricted radius of the recording tip. The ground electrode drains off currents originating from electromagnetic interference. Needle electrodes currently available include monopolar, standard or coaxial concentric, bipolar concentric, single fiber, multielectrode, flexible wire electrodes, and glass microelectrodes.

Monopolar and concentric needle electrodes are most commonly used. The monopolar needle is made of stainless steel and coated with Teflon except at the tip. It is used with a surface reference and ground electrode. It is less painful, less expensive, and records a larger action potential than the concentric needle. The CMUAP amplitude recorded with a faulty monopolar needle is decreased, possibly because the increased surface area of the recording tip has a decreased impedance and expands the region over which potentials are averaged.[172] The duration of the CMUAPs recorded is also decreased when using a monopolar needle with a defective Teflon coating.[171] Monopolar needle impedance is lower than that of concentric needles. Under certain circumstances, it may be advantageous to use a monopolar needle for stimulation of a nerve. Care must be taken to strip back the Teflon coating several millimeters, otherwise the concentrated focus of stimulation produced by a normal needle may cause excessively high local electrical current flow, resulting in focal nerve injury.[171] Alternatively, the use of an unmodified monopolar needle for stimulation with a current duration limited to 0.05 ms reportedly prevents axonal injury.[173,174]

The coaxial concentric needle has a stainless steel cannula with a nichrome, silver, or platinum wire in the center of the shaft. The central wire electrode is separated from the cannula by an insulating material. The needle is designed to register a potential difference between the center wire and the shaft. A separate surface electrode serves as the ground.

The bipolar concentric needle has a cannula which contains two fine stainless steel or platinum wires. This needle is larger in diameter than the coaxial concentric needle. The potential difference between the two inner wires is recorded. The cannula serves as the ground. Thus, electrical activity is recorded from a substantially smaller area than that recorded with the standard concentric needle.

The concentric needle electrodes have increased common mode rejection and record a slightly reduced action potential amplitude relative to the monopolar electrode since the cannula, which serves as a reference, detects similar activity to the active recording surface. The concentric needles also register slightly smaller motor unit amplitudes compared to monopolar needles because there are fewer muscle fibers within the recording range of the active electrode. The concentric needle electrodes generally produce a more stable baseline than that achieved with monopolar electrodes.

The single fiber needle electrode has a significantly smaller leading tip from which to record muscle activity. It specifically records action potentials from individual muscle fibers rather than motor units. It typically has a cannula diameter of 0.5 mm or less and the active recording wire is 25 μm in diameter. The wire is located proximal and opposite to the bevel of the needle. The cannula acts as the reference and a separate surface ground electrode is necessary. Usually only one, but sometimes two muscle fiber action potentials are detectable at any given time with this type of electrode. Monopolar or concentric needles, however, may record 10 or more muscle fibers.[172]

Electromyography

EMG is a means of assessing muscle action potentials using a needle electrode in the extracellular space. The needle examination can be broken down into four separate procedures, each of which conveys significant information regarding the in-

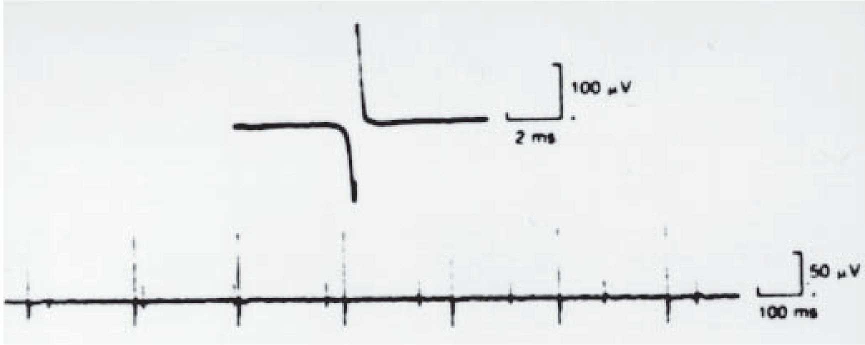


Fig. 9-17. Fibrillation potential. Electrical activity associated with spontaneously contracting muscle fiber. The potentials usually fire at a constant rate. Amplitudes range from 25 µV to 200 µV; durations extend from 0.5 ms to 2.0 ms. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 655.

tegrity of the motor unit: (1) insertional activity, (2) examination at rest, (3) examination at minimal exertion, and (4) examination at maximal exertion.

Insertional Activity

The passage of a needle electrode through a muscle membrane creates a brief electrical discharge that is both visible and audible with appropriate amplification. Insertional activity that is decreased or absent indirectly indicates a decrease in the number of muscle fibers secondary to severe atrophy or fibrosis, or both, of the muscle tissue. If insertional activity is increased, specifically if it lasts longer than the cessation of needle movement in the muscle, this indirectly reflects muscle membrane instability. Increased insertional activity may be found in the presence of a denervation process, myotonic disorder, or myopathic disease.

Examination at Rest

A normal muscle at rest is electrically silent. If electrical activity occurs at rest, it is referred to as spontaneous activity and generally reflects a pathological condition. Fibrillations and positive sharp waves are examples of spontaneous potentials and reflect abnormal discharges originating from single muscle fibers.

Spontaneous activity. Spontaneous activity is the electrical response found after needle electrode insertion activity and volitional muscle contraction has ceased. Spontaneous activity is not typically found in normal muscle at rest; it is also not present in muscles with isolated disuse atrophy; it can occur in paraspinal muscles following myelography, lumbar puncture, surgical intervention, or local trauma. It also may occur in the weak limbs of patients with upper motor neuron lesions. It is present in some myopathic disorders but most often in denervated muscle. Spontaneous activity may present as fibrillations, positive sharp waves, fasciculation potentials, myokymic discharges, or complex repetitive discharges (Figures 9-17 through 9-21).

Fibrillations are the result of spontaneous depolarizing single muscle fibers. They range in amplitude from 25 to 200 µV and in duration from 0.5 to 2.0 ms. They usually have a triphasic morphology and occur at a regular rate.

Positive sharp waves also represent a form of spontaneous activity. They are characterized by a wave with an initial positive deflection followed by more gradual return to baseline. They generally occur at a regular rate and, like fibrillations, are suggestive of

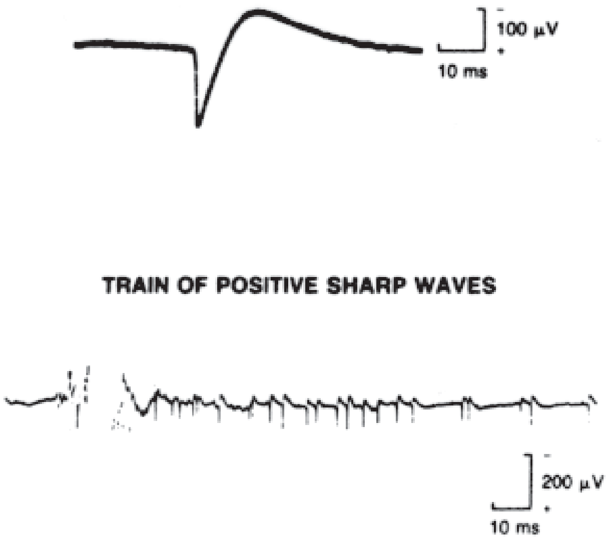


Fig. 9-18. Positive sharp wave. The potential is biphasic with an initial rapid positive deflection. They fire at a constant rate. Amplitudes are up to 1 mv. Duration ranges from 10 to 1,000 ms. Positive sharp waves are not specific for muscle fiber damage. Motor unit action potentials and myotonic discharges may have the same configuration. They do not however, fire at a constant rate. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 656.

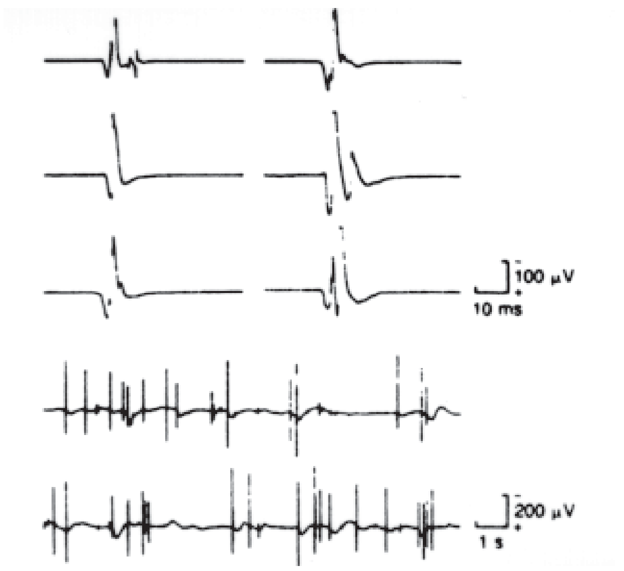


Fig. 9-19. Fasciculation potentials. Often associated with visible fasciculations in the limb, fasciculations have the same configuration as motor unit potentials but fire spontaneously. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 659.

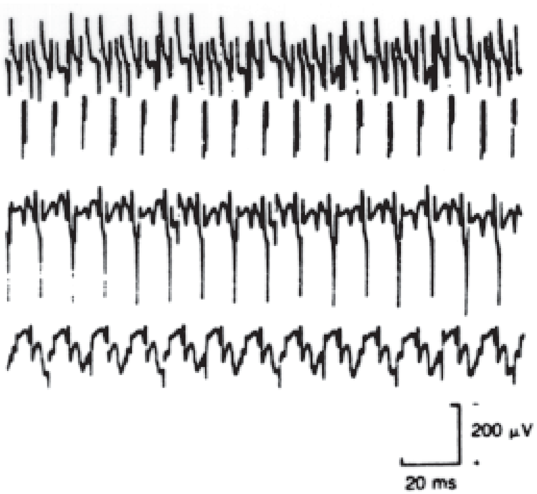


Fig. 9-21. Complex repetitive discharges. A polyphasic action potential that begins spontaneously or after needle movement. They have uniform frequency, shape, and amplitude with abrupt onset and cessation. Amplitudes range from 100 µV to 1 mV. Discharge frequency ranges between 5 Hz and 100 Hz. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 658.

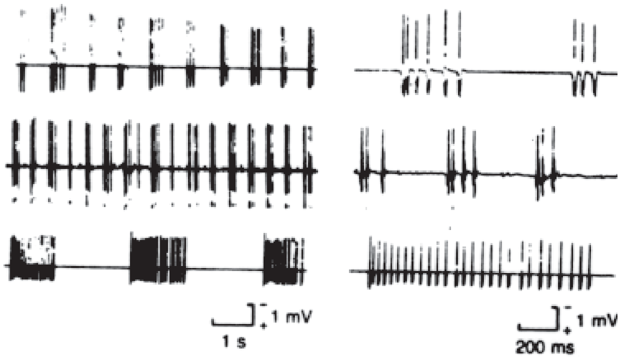


Fig. 9-20. Myokymic discharges. Commonly, discharges are brief, repetitive firings of single motor units for a few seconds at a uniform rate ranging from 2 Hz to 60 Hz. The sequence may be repeated for the same potential. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 660.

TABLE 9-7

QUANTIFICATION OF FIBRILLATION POTENTIALS AND POSITIVE SHARP WAVES

Grade	Definition
1+	Transient but reproducible runs of fibrillation potentials or positive sharp waves in two different sites
2+	Transient but reproducible runs of fibrillation potentials or positive sharp waves in more than two sites
3+	Spontaneous activity at rest in at least two different sites
4+	Profound spontaneous activity, which may fill the screen

Source: Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 2nd ed. Philadelphia, Pa: FA Davis; 1989: 219.

an unstable muscle membrane. These waves are seen in neuropathic, some myopathic, and even some central nervous system disease (Table 9-7).

Spontaneous activity typically occurs within 2 to 3 weeks after nerve insult. Muscle fiber sensitivity to acetylcholine increases a hundred-fold after denervation and is known as denervation hypersensitivity,[175,176] which possibly contributes to the generation of spontaneous activity, but is not felt to be the sole or even the primary factor.

Examination at Minimal Contraction

Motor units have a distinct appearance, indicative of the extracellular recording of action potentials as they move toward and away from the active and reference electrodes. Specific attributes provide important information regarding particular pathologic processes. Technical factors, including the type of needle electrode employed, the muscle being studied, the temperature, and age of the patient may all influence aspects of motor unit morphology (Figure 9-22).

Motor Unit Electrical Morphology

***Duration*.** The duration of the unit activity is the measurement from the onset of the potential as it leaves the baseline to its final return to baseline.[177] It normally varies from 5 to 15 ms when concentric needle electrodes are used.[160] Different durations will be recorded from the same motor unit depending on the type of electrode used. Duration will be longer with monopolar needles. The use of a surface reference electrode decreases the common mode rejection and more low frequency activity is included. A ratio of monopolar to concentric durations has been documented as 1.86:1.[178] Motor unit duration increases by 10% for each 1°C decrease in temperature between 37°C and 30°C. A 30% increase occurs for each 1ºC decrease between 30°C and 20°C.[179]

Phases. The shape of the normal unit recording outside the endplate region has a triphasic appearance. There is an initial positive deflection followed by a negative spike and then another positive deflection before returning to baseline. The section of the motor unit recording between two baseline crossings is termed a phase. Adding one to the number of baseline crossings is one method of calculating the number of phases comprising a motor unit potential.[177]

Polyphasic motor units have more than four phases and reflect a relative asynchrony of muscle fiber firing within the motor unit. According to Kimura,[160] 5% to 15% of motor units may be polyphasic in normal muscles. The acceptable percentage of polyphasic units varies between muscles and with the age of the patient. An extensive compilation has been tabulated and can be found in Chu-Andrews and Johnson's text.[180] A drop in temperature to 25ºC will increase the percentage of polyphasic potentials by 10%.[181]

Long duration polyphasic motor unit potentials suggest a significant loss of synchrony and can be

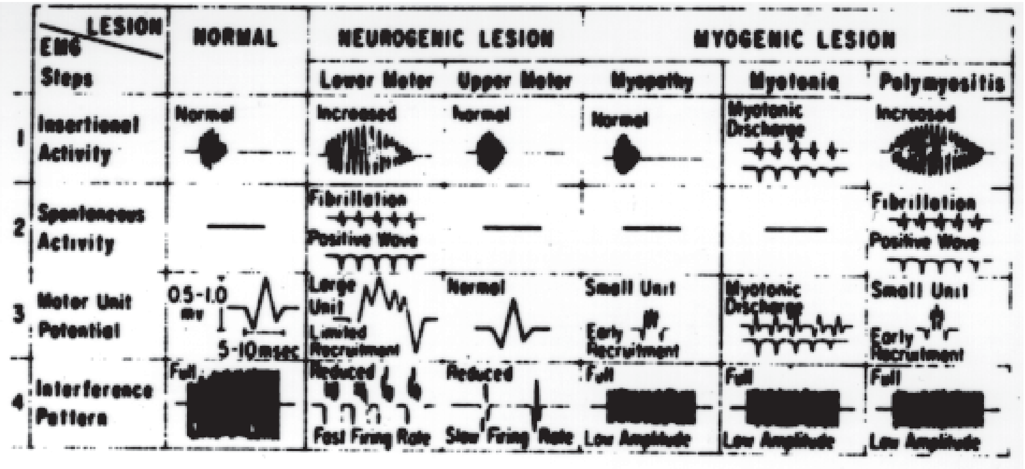

LESION
EMG Steps
NORMAL
NEUROGENIC LESION
MYOGENIC LESION
Lower Motor
Upper Motor
Myopathy
Myotonia
Polymyositis
1 Insertional Activity
Normal
Increased
Normal
Normal
Myotonic Discharge
Increased
2 Spontaneous Activity
Fibrillation
Positive Wave
Fibrillation
Positive Wave
3 Motor Unit Potential
0.5-1.0 mv
5-10msec
Large Unit
Limited Recruitment
Normal
Small Unit
Early Recruitment
Myotonic Discharge
Small Unit
Early Recruitment
4 Interference Pattern
Full
Reduced
Fast Firing Rate
Reduced
Slow Firing Rate
Full
Low Amplitude
Full
Low Amplitude
Full
Low Amplitude

Fig. 9-22. Synopsis of electromyographic findings, including insertional activity, spontaneous activity, motor unit potential morphology, and interference pattern observed normally as well as with neurogenic and myogenic pathology. Adapted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1989: 252.

seen during lower motor neuron degeneration and regeneration. Short duration polyphasic potentials are indicative of muscle fiber loss and are classically seen in myopathic processes or during early reinnervation following axonal loss.

Amplitude. The amplitude of the motor unit is the summation of muscle fiber activity within a 1- mm radius from the electrode. Thus, the distance of the tip of the electrode from a group of muscle fibers drastically changes the amplitude of the motor unit potential. To prevent fallacious measurement of potentials too distant to the electrode, motor units potentials suitable for assessment should have a rise time of less than 500 µs and preferably 100 to 200 µs.[160] The rise time is defined as the time elapsed between the initial positive peak to the next negative peak.[177] During minimal contraction, amplitudes range from several hundred microvolts to a few millivolts. During maximal contraction, normal values range from 2 mV to 8 mV. Occasionally, muscles of the hands may generate amplitudes as high as 12 mV and still be considered within the normal range.[180] Amplitudes of motor unit potentials examined with a monopolar needle are greater than those measured by concentric needles. Comparisons documented at minimal contraction measured the mean ratio of amplitudes recorded from monopolar to concentric electrodes at 2.05:1.[180] Temperature differences will also alter motor unit potential amplitudes. Differential slowing of conduction along the fibers summating to generate the potential causes temporal dispersion. Mean amplitudes will decrease by a factor of 2% to 5% for every 1°C decrease in temperature.[181]

Large amplitude motor unit recordings suggest prior lower motor neuron injury. Reinnervation and local sprouting concentrate the muscle fibers comprising the motor unit within a smaller region. Consequently, a larger number of muscle fibers are located near the tip of the electrode and a more significant summation is recorded. Small amplitude motor units are observed when muscle fibers are lost (eg, myopathic processes) or during very early reinnervation after a lower motor neuron injury (Figure 9-23).

Evaluation of motor unit electrical morphology is an indispensable component of the electromyographic evaluation. In situations where quantitative analysis is necessary, strong consideration should be given to use of concentric needle electrodes. Most reference values, including duration, amplitude, shape, and rise time, were established utilizing concentric needles. The use of a surface reference electrode during monopolar needle studies inherently leads to wide variability among the variables examined. Subtle differences in surface reference electrode placement may drastically change the common mode rejection and ultimately the duration and amplitude of the motor unit potentials.

Recruitment

The sole purpose of motor unit recruitment is to generate very specific amounts of force smoothly through muscle contraction. The process follows Henneman's size principle.[163] Small, low-tension units fire initially. As greater amounts of force are generated, larger, faster units also begin to fire. Force generation is the product of an increase in the firing rate of individual motor units and the activation of additional units. Normally, motor unit firing plateaus and the primary mode of increasing force is the recruitment of more motor units.

Assessment of the recruitment rate is an essential technique, allowing for the evaluation of motor unit pathology. First recruited motor units begin firing when contraction levels are barely percep-

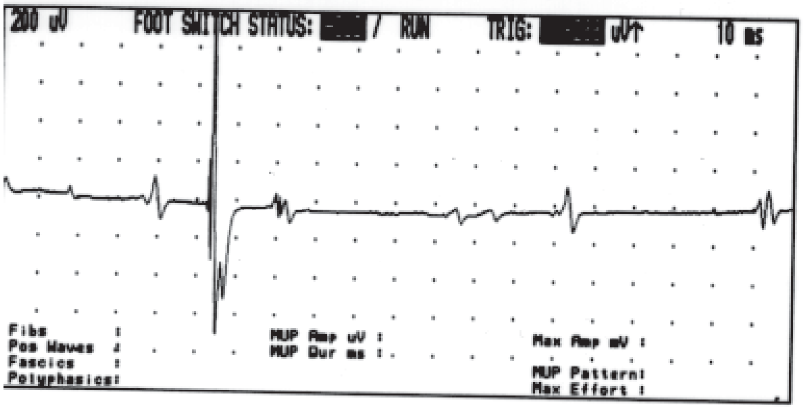


Fig. 9-23. Electromyographic display of a large amplitude polyphasic motor unit potential with associated satellite potential. The entire potential is of long duration. The satellite is not included in the measurement of duration. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

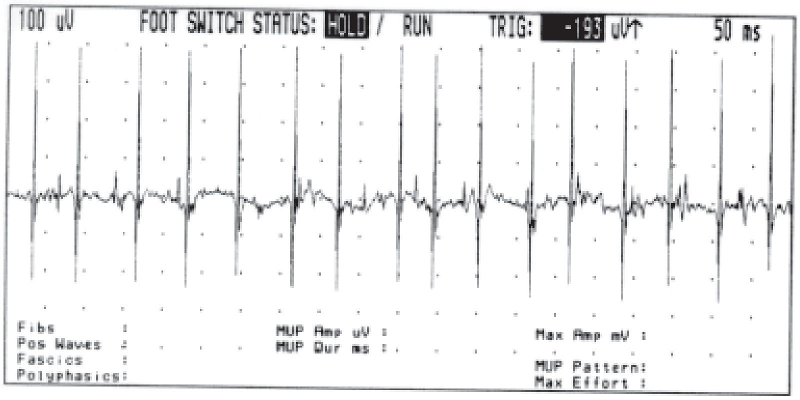


Fig 9-24. Recruitment rate of a first recruited motor unit firing at 16 Hz. This finding may be the earliest suggestion of motor unit pathology. It may occur with axonal loss or conduction block. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

tible. The frequency at which the unit is firing, termed the recruitment rate, is semirhythmic but becomes relatively stable just before the next motor unit begins to fire. Normal firing frequencies range from 5 to 15 Hz (Figure 9-24). On occasion, it is difficult for patients to contract at low enough levels to recruit less than three motor units. An alternative method of describing recruitment is the recruitment ratio. The rate of firing of the fastest unit is divided by the number of units firing. Normal ratios are around 5. A ratio greater than 10 is abnormal.[177]

Recruitment abnormalities may be the earliest evidence of motor unit pathology observed during the electrodiagnostic examination. Motor neuron loss is exhibited as an increase in the firing frequency. As fewer units are available to be recruited, adequate force generation is achieved through higher rates of firing by intact units.[182] Loss of muscle fibers creates a different recruitment pattern. Motor units are intact, yet, because a smaller number of muscle fibers are available, a greater number of normally firing motor units are recruited earlier to attempt adequate force production. The term rapid or early recruitment is used to describe this situation but the term is easily misunderstood. Rapid recruitment conveys only that the timeframe in which many motor units are normally recruited is compressed. The recruitment frequency is normal (ie, recruitment of the second unit does not occur when the first unit is firing at less than 5 to 15 Hz.[177]

Examination at Maximal Contraction

Assessment of motor unit firing during maximum voluntary contraction provides a gross assessment of motor unit integrity.

The interference pattern is a gestalt estimate of the total number of motor units activated per second during maximal contraction.[182] In a healthy individual providing maximal effort, individual motor unit potentials cannot be discerned because the multitude of units firing at upwards of 50 Hz fill the screen and is deemed a full interference pattern. When motor neurons are lost, fewer motor unit potentials are available to summate and the patient will not be able to produce numbers sufficient to fill the screen. The interference pattern is said to be reduced. Individual or discrete motor unit potentials may be observed when axonal injury is severe.[180]

The interference pattern associated with the loss of muscle fibers has been described previously. Low amplitude motor units fill the screen during only a minimal voluntary contraction.

The utility of interference pattern interpretation may be confounded by several variables. A normal patient may not produce a maximal contraction because of pain, poor cooperation, or poor coordination. Patients with severe sensory neuropathies may not be able to gauge the intensity of their muscle contractions due to loss of proprioceptive feedback. Upper motor neuron abnormalities such as spinal cord injury or stroke will also limit the patient's ability to generate a maximal contraction. Incomplete conduction blocks may produce discrete interference patterns and be misinterpreted as severe axon loss if not interpreted in conjuction with the rest of the electrodiagnostic examination.

Nerve Conduction Studies

Conduction velocity may be determined in either motor or sensory nerves. Although some nerves are not readily accessible to study, a large number, including cranial nerves and upper and lower extremity nerves, are available for testing. Electrical stimulation of a sensory or motor nerve results in a recordable action potential. The potential is a summated response of the nerve fibers stimulated. In the case of motor NCSs, motor nerve fibers of a selected nerve are stimulated and the summated electrical response (CMAP) is picked up over a muscle innervated by that nerve. In sensory NCSs, sensory fibers of a selected nerve are stimulated at a point along the length of the nerve. An action potential, which represents the sum of the action potentials generated by these sensory fibers, is obtained with electrode pickup at another site along the course of the nerve.

Latency

Latency is the time in milliseconds between application of the nerve stimulus and the onset of the recorded evoked compound action potential. Thus, latency recordings reflect the speed of the fastest conducting fibers within a nerve. A latency is usually obtained with the most distal stimulation along a nerve. Latencies obtained from more proximal portions of the nerve may be erroneous due to changes in the fascicular composition of the nerve.

Conduction Velocity

Conduction velocity is determined by obtaining latencies from a distal and a proximal stimulation site. It is specifically calculated by dividing the distance between the two sites by the latency difference between the proximal and distal sites:

$$\frac{\text{Distance}}{\text{Time}} = \frac{\text{PD} - \text{DD (mm)}}{\text{PL} - \text{DL (ms)}} = \text{Conduction Velocity(m/s)}$$

where PL is proximal latency, DL is distal latency, PD is distance from site of proximal stimulation to recording electrode, and DD is distance from site of distal stimulation to the recording electrode.

Amplitude

Amplitude of a CMAP is primarily dependent on the number and density of innervated muscle fibers and their synchrony of firing. The integrity of the neuromuscular junction also affects the amplitude of the motor action potential. The SNAP amplitude is primarily dependent on the number of functioning large myelinated axons present.[183]

The amplitude of the CMAP and SNAP are also dependent on the distance between the site of stimulation and the actual position of the nerve, as well as on the intensity of stimulation. The greater the soft tissue thickness, the greater the distance between stimulation site and nerve, which frequently results in attenuation of the action potential amplitude due to incomplete nerve stimulation. If the recording electrode is not close to the nerve or muscle due to fat or other tissue, the signal will also be attenuated.

Duration

Duration of an action potential reflects the degree of variation of latencies between the fastest and slowest conducting fibers. Abnormal temporal dispersion, a reflection of dyssynchrony of conduction, occurs when there is an excessive difference between the fastest and slowest fibers. This finding is typically accompanied by a concomitant reduction in amplitude. If some of the nerve fibers are actually blocked rather than merely slowed by a disease process or by focal injury, the area of the action potential will also be reduced.

Utility of Electrodiagnostic Studies

The potential use of electrodiagnostic techniques to determine the location and severity of nerve injuries has been postulated as far back as World War I.[4] It has only been since the 1970s, however, that the equipment and general expertise in the field have improved to levels at which sensitive information might be provided consistently and reliably. Currently, electrodiagnostic techniques are the most objective and quantitative means of assessing nerve injuries.[184] The complex nature of nerve injuries and the subtleties of interpreting information derived from electrodiagnostic studies require that a physician with special training in the diagnosis and treatment of neuromuscular disorders plan, administer, and interpret such studies. Extensive training in neurophysiology and electrophysiologic techniques is essential to ensure that correct and appropriate diagnoses are obtained by the electrodiagnostic medical consultant.

Determination of Severity

The primary determinant of recovery following nerve injury is the severity of the injury. The predominant electrodiagnostic features observed during Wallerian degeneration are the progressive reduction in the amplitude of evoked potentials along the entire length of the nerve and the appearance of denervation potentials in affected muscles.[45] The electrodiagnostic findings observed following nerve injury are dependent on three main variables: (1) the time elapsed from the injury, (2) the type of nerve injured, and (3) the length of the injured nerve distal to the focus of injury.

Following transection of a nerve, CMAPs and SNAPs cannot be elicited by stimulation of the nerve proximal to the focus of injury. Stimulation distal to the injury, however, will result in an action potential during a short period until axonal degeneration occurs. In humans, the ability to evoke a distal CMAP begins to wane 3 to 6 days following injury. In a recent study by Chaudhry et al,[45] an ulnar nerve injury at the elbow showed an amplitude drop of 10% at day 3 postinjury, a 90% drop by day 6, and loss of the ability to evoke an action potential at day 9. Diminution of SNAP amplitudes following axonal injury lag behind the loss of CMAP amplitudes by 2 to 3 days. On average, the ability to evoke a SNAP is lost between 9 and 11 days following injury. Following transection of the sural nerve at the calf, a SNAP amplitude drop of 20% was first noted at postinjury day 5, a 60% drop by day 9, and loss of the ability to evoke a SNAP at day 11.[45] The early failure of motor neurons to propagate an evoked potential distal to the site of injury is the electrophysiologic correlate of neuromuscular junction failure. The neuromuscular junction fails prior to axonal degeneration.[37]

The length of the distal stump also has a profound effect on the timing of the loss of ability to evoke an action potential. While the exact mechanism remains unclear, trophic support for the portion of the nerve distal to the focus of injury remains intact for a period of time following injury and is directly proportional to the length of the nerve distal to the focus of injury. Thus, the ability to generate an action potential distal to the site of injury will last for a longer period of time when the distal portion of the nerve is longer. For example, an injury to the ulnar nerve at the elbow with a length distal to the injury of 25 cm revealed a CMAP amplitude drop of 80% by day 6. An injury to the facial nerve, with a length distal to the site of injury of only 12 cm showed an 80% CMAP amplitude drop by day 3.[185] Injuries at more proximal regions along similar nerves will also show the same time differences. An ulnar nerve injury in the arm with a length of 45 cm distal to the focus of the injury may reveal a CMAP amplitude drop of only 30% by day 6 and not be lost until the 11th or 12th day. Electromyographic abnormalities suggestive of axonal injury also lag behind the onset of injury and are related to the length of the nerve distal to the site of injury. The onset of spontaneous activity closely parallels the loss of ability to evoke a SNAP.[184] For example, spontaneous activity may be seen as early as 7 days in facial muscles following axonal injury to the facial nerve. On the other hand, a very proximal injury at the root or plexus level may not manifest evidence of denervation by electromyographic evaluation for upwards of 21 days.

The timing of electrodiagnostic evaluations following nerve injuries may have a profound impact on the interpretation of the study. If NCSs are performed too soon, the ability to elicit a distal action potential may be misinterpreted as a less severe injury such as conduction block (see below) rather than an axonal injury. Inaccurate localization may also occur. If studies are performed during the window when the CMAP is lost but the SNAP is still obtainable, the findings may be interpreted to suggest that a lesion proximal to the dorsal root ganglion is present. A study performed later may reveal that the ability to evoke a SNAP has also been lost, supporting that the location of the injury is distal to the dorsal root ganglion, which is prognostically better than the more proximal lesion and may be amenable to surgical intervention.

One of the major applications of electrodiagnostic medicine is the quantification of each physiologic process that underlies the clinical manifestation of nerve injury. During early evaluations, physical examination tends to overcall the severity of an injury. Late in the course of reinnervation, trick movements may be learned by the patient as compensatory methods of overcoming persistent weakness. In this situation, findings on the physical examination can be misconstrued as greater return of neurologic function than is truly present.

As previously noted by Seddon,[31] it is extremely common to have the coexistence of axonal degeneration, conduction block, and conduction slowing with temporal dispersion following acquired nerve injuries. Analysis of nerve conduction parameters in conjunction with electromyographic findings allows the character of the injury to be determined. Following the initial delay, axonal injury leads to a drop in the amplitude of the evoked action potential along the entire length of the nerve (ie, during stimulation both proximal and distal to the focus of injury). The duration of the potential changes little. The area under the curve is also reduced, reflecting the loss of fibers adding to the summated action potential.

Conduction block is evidenced by a drop in the amplitude of the action potential during stimulation proximal to the site of injury when compared

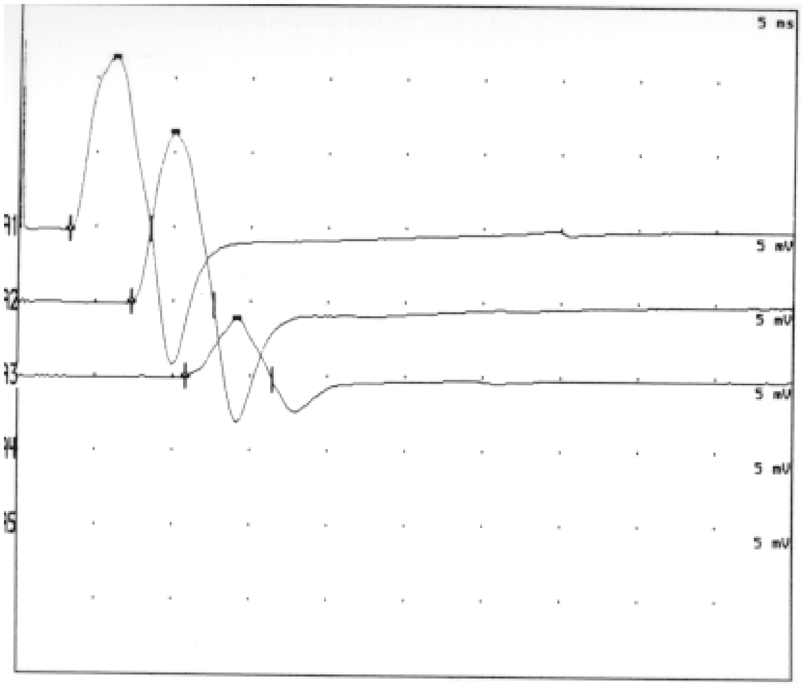


Fig 9-25. Nerve conduction study of the ulnar nerve revealing conduction block at the elbow. Note that both the amplitude and area are markedly decreased with little evidence of temporal dispersion or change in duration, compared to more distally evoked compound motor unit action potentials. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

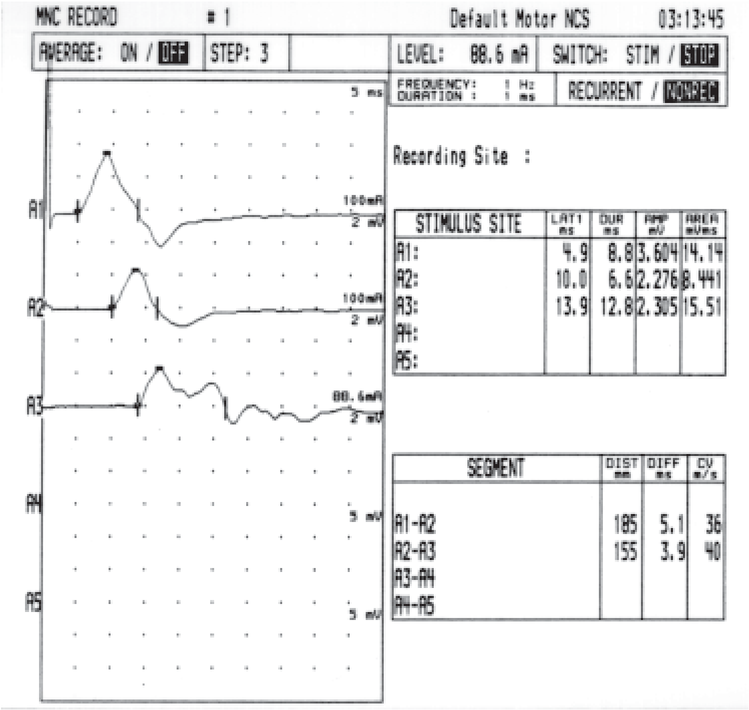
to stimulation distal to the focus. A comparison of distal CMAP amplitude to proximal CMAP amplitude revealing an amplitude drop of greater than 20% has been suggested to be significant[186] (Figure 9-25). In pure conduction block, the duration of the potential does not change appreciably. Thus, a reduction in amplitude and area are observed.

Conduction slowing with temporal dispersion is the third type of injury that can be discerned. Conduction slowing with temporal dispersion suggests that not only the fastest fibers have been injured but all types of fibers. Differences in the speed of conduction increases the duration of the action potential. The decreased synchrony of firing, manifested by spreading of nerve fiber potentials over a greater timeframe, is reflected in a decrease in the amplitude of the potential as the potentials do not summate. The area under the curve, however, is not decreased because the number of fibers activated is relatively normal (Figure 9-26). Conduction slowing with temporal dispersion may be commonly seen during more insidious, cumulative types of trauma. It may also be seen during reinnervation, whether spontaneous or following surgical nerve repair.

Conduction block and conduction slowing with temporal dispersion are manifestations of demyelinating, neuropraxic injuries. Thus, electromyographic evaluations should not reveal spontaneous activity (Table 9-8). The reader is referred to the discussion on peroneal neuropathies for a clinical example of the electrodiagnostic determination of nerve injury severity.

Localization

The ability to localize nerve injuries requires an in-depth knowledge of neuroanatomy. Comprehensive understanding of dermatomal and myotomal distributions, anomalous innervations, innervations of specific muscles including nerve branch, nerve trunk, course through the plexus of origin, and nerve roots is essential for accurate localization. Electrodiagnostic localization may be especially helpful in combat-related injuries where injuries may occur practically anywhere along the length of the nerve and at multiple sites. Accurate localization also conveys the extent of injury, which may reflect functional recovery and prognosis. Among the factors that affect the results of both natural and surgical recovery is the location of the injury. More distal lesions are more likely to recover in comparison to more proximal injuries. Different nerves also



MNC RECORD # 1 Default Motor NCS 03:13:45

AVERAGE: ON / OFF STEP: 3 LEVEL: 88.6 mA SWITCH: STIM / STOP

FREQUENCY: 1 Hz
DURATION : 1 ms

RECURRENT / NONREC

Recording Site :

STIMULUS SITE	LAT1 ms	DUR ms	AMP mV	AREA mVms
A1:	4.9	8.8	3.604	14.14
A2:	10.0	6.6	2.276	8.441
A3:	13.9	12.8	2.305	15.51
A4:				
A5:				

SEGMENT	DIST mm	DIFF ms	CV m/s
A1-A2	185	5.1	36
A2-A3	155	3.9	40
A3-A4			
A4-A5			

Fig 9-26. Nerve conduction study of the ulnar nerve revealing conduction slowing and temporal dispersion at the elbow. Note that while the amplitude is decreased compared to distally evoked compound motor unit action potentials (CMUAPs), the duration is increased and the area has remained essentially stable. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

TABLE 9-8

ELECTRODIAGNOSTIC PARAMETERS SUGGESTING SEVERITY OF NERVE INJURY

	Axon Loss	Temporal Dispersion	Conduction Block
Nerve Condution Study Parameters*			
Duration	No change	Increased	No change
Amplitude	Decreased	Decreased	Decreased
Area	Decreased	No change	Decreased
Electromyographic Study Parameters			
Spontaneous activity	Present	Absent	Absent
Reduced recruitment	Present	Possible	Present

*Comparison of proximal to distal compound motor action potentials (CMAPs). An increase in duration of greater than 15% and a reduction in amplitude of greater than 20% are considered abnormal when comparing proximal to distal CMAPs.

respond and repair differently when subjected to similar injuries. Radial nerves are reputed[187] to have the best recovery, followed by median and ulnar, and all usually fare better than injuries to nerves in the lower extremities. Lastly, recovery from injury to pure motor or sensory nerves is commonly better than from injuries to mixed nerves. The principles of localization, presented later in this chapter, dealing with more common areas of entrapment injuries can be readily applied to combat-related injuries.

Nerve conduction studies can be employed to differentiate nerve root lesions from more distal injuries. A detailed discussion can be found in the section concerning brachial plexus injuries. Conduction studies are also helpful to localize focal areas of conduction block or conduction delay. The section on ulnar nerve injuries provides a paradigm, including short segment incremental studies and the use of conductions along several different branches, that can be applied in the unique situations encountered with traumatic injuries. Involvement of multiple nerves, even those not clinically suspected by physical examination, can be elucidated by NCSs.

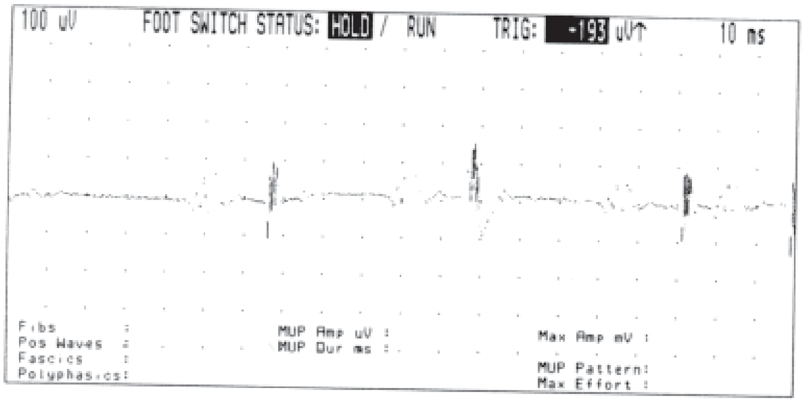


Fig 9-27. Electromyographic display of a low amplitude, long duration polyphasic motor unit potential. The presence of such a motor unit potential may suggest early reinnervation following peripheral nerve injury. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

Electromyographic techniques are especially helpful when the injury primarily involves the loss of axons. Evidence of denervation observed in muscles distal to the lesion with sparing of muscles innervated by the same nerve but proximal to the lesion help to strategically place the lesion. Examples of this can be found in sections discussing the differentiation of radial nerve from posterior interosseus nerve injuries and median nerve from anterior interosseus nerve injuries.

Monitoring Recovery

Electrodiagnostic studies can be used to follow both natural and postsurgical reinnervation. While reinnervation occurs through both collateral sprouting and regeneration, it has been suggested by Buchthal and Kuhl[188] that, in humans, the former is more important. Early evidence of reinnervation can be seen on EMG. Voluntary motor unit activity was observed[189] following end-to-end anastomoses 4 to 7 months after suture. Electromyographic activity was detected after 12 months when grafting was warranted.[189] When a partial injury has been sustained, voluntary motor unit potentials may be evident within several weeks after injury. Low amplitude, polyphasic, long duration motor unit potentials are seen initially and may predate the loss of denervation potentials (Figure 9-27). Single fiber EMG analysis provides a useful technique to explore the earliest signs of reinnervation. Locking one single fiber potential in time (triggering) allows for the assessment of the latency difference between the triggered single fiber action potential and the other single fiber action potential in the motor unit. This latency difference is termed the inter-potential interval (IPI). The tenuous nature of the neuromuscular connection in an immature motor unit is suggested by two findings. *Blocking* occurs when neuromuscular transmission fails intermittently and the action potential of the single motor fiber is not generated. *Jitter* is the variation of the IPIs. A small amount of jitter is seen in normal motor units. Jitter is increased in immature motor units due to fluctuations in the time needed for summation of endplate potential to evoke an action potential at the neuromuscular junction. As the new connections mature, conduction along the sprouted fibers will increase and the potential will become incorporated in the parent motor unit potential (Figure 9-28). Early on, the duration of the motor unit potential will be increased, but as reinnervation proceeds the duration will approach normal and the polyphasic nature of the unit will decrease. Summation will improve as the fibers fire more synchronously. As more fibers are now incorporated in the motor unit, the amplitude may become exceptionally large (Figure 9-29).

Nerve conduction studies may also be used to chart the course of recovery. Very low amplitude, temporally dispersed action potentials may be seen during the initial stages of reinnervation. Conduction velocity across the affected area may be exceedingly slow, in some cases less than 10 m/s. It has been estimated by Buchthal and Kuhl[188] that the action potentials of at least 40 fibers of greater than 7 μm in diameter need to summate before a compound potential of 0.02 μV with a conduction velocity of 10 m/s can be generated. This is felt to be the minimum parameters required to distinguish the potential electrodiagnostically from background noise. While conduction velocity increases and the amplitudes of evoked sensory and motor potentials improve, they commonly do not return to baseline values. Hodes et al[190] observed that even as far out as 12 to 42 months following partial transection, conduction velocities continued to range between 40% to 60% of normal. Similar findings were observed by Cragg and Thomas.[191] Conduction velocities plateaued at 75% of normal at 12 months following injury. Buchthal and Kuhl[188] and Donoso et

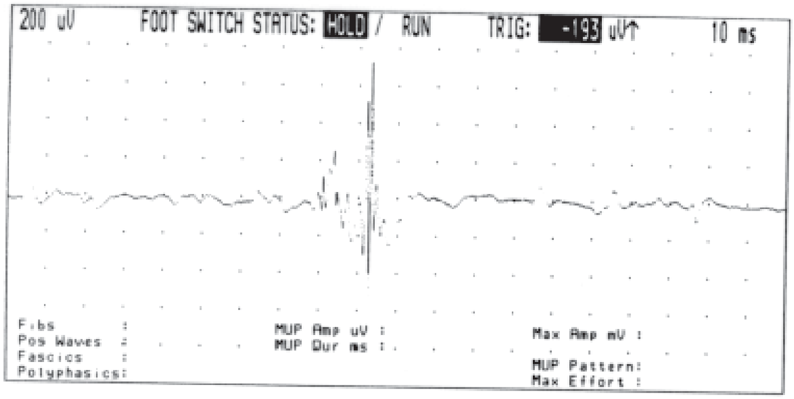


Fig 9-28. Electromyographic display of a normal amplitude, long duration polyphasic motor unit potential commonly observed as reinnervation becomes more stable and synchronization of muscle fiber firing improves. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

al[189] both observed the return of CMAPs 4 to 7 months following nerve suturing. Distal motor latencies improved over time but rarely returned to normal range. SNAPs were first obtainable in Buchthal and Kuhl's[188] study 4 months postsuturing and 7 months following nerve grafting. Only 54% of the Donoso et al[189] groups had detectable SNAPs as far out as 20 months. In both studies, even when SNAPs could be evoked, amplitudes rarely improved to better than 10% of normal.

While these data are intriguing, they become more important when related to the return of sensory and motor function. Return of sensory function does not correlate well with SNAP amplitude changes. According to Buchthal and Kuhl,[188] the ability to perceive tactile stimulation returns when approximately 1% of myelinated sensory fibers reinnervate. This correlated to 5 to 7 months after suturing when it did occur. After 28 months, tactile perception had improved; light touch could be discriminated from pinprick, but the site of stimulation could not be discerned. Three years following suturing, the patient began to localize stimuli. The lack of correlation between SNAP amplitude and return of sensibility can be explained by the temporal dispersion that persists after sensory nerve injury. Also, sensory potentials measure only the fastest and largest myelinated fibers. Thus, sensibility conveyed along small and unmyelinated fibers will not be included in the SNAP. As techniques become more refined in the future, it may be possible to monitor the area of the SNAP, which may better correlate with the return of sensory function.

In contrast to sensory function, a strong correlation between motor function and the amplitude of the CMAP has been determined. Donoso et al[189] observed that a return of CMAP amplitude to over 40% of normal correlated with a return of motor power in the affected muscles to good (4/5) or normal (5/5) strength. The ability of the reinnervated muscle to generate near normal power when fewer than normal CMAPs are obtained and discrete recruitment patterns are observed on EMG, reflects the remarkable resilience of the motor unit. So long as 50% to 75% of motor neurons remain intact, functional recovery may be achievable through collateral sprouting and regeneration.[184]

Limitations of Electrodiagnostic Studies

Nerve conduction studies and EMG convey electrophysiologic abnormalities reflecting periph-

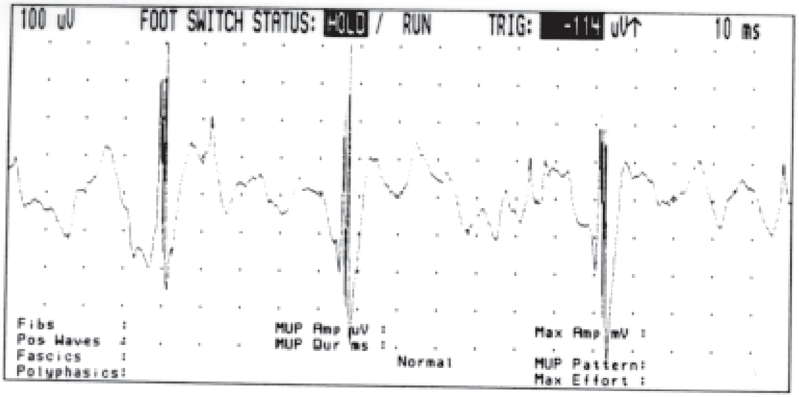


Fig 9-29. Electromyographic display of a normal amplitude, normal duration polyphasic motor unit potential. This type of unit potential may suggest old stable reinnervation. Commonly, as summation improves, these potentials may be of large amplitude. Photograph courtesy of MAJ Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

eral nerve pathology. It should be understood, however, that these studies cannot be used to infer precise pathological changes (eg, findings may suggest axonal injury but ischemic axonal injury cannot be differentiated from stretch-related axonal injury).

Subtle differences in severity or improvement cannot truly be differentiated by electrodiagnostic studies. Findings suggestive of demyelination can be discerned from those consistent with axonal injury, and to some extent the relative roles of these processes can be inferred. Yet, the hierarchical levels of axonal pathology as outlined by Sunderland (second to fifth degrees) cannot be truly appreciated electrodiagnostically. The ability to evoke an action potential during stimulation proximal to an injury does convey the integrity of the nerve. After an appropriate time delay, action potentials evoked distal to the lesion also reflect continuity. The inability to obtain action potentials does not confer the loss of anatomic continuity. Thus, while electrodiagnostic findings may suggest a very severe axonal injury, they cannot be used to conclude or confirm complete disruption of the nerve.

REHABILITATIVE MANAGEMENT OF PERIPHERAL NERVE INJURIES

General Principles

Rehabilitation of peripheral nerve injuries is absolutely essential to ensure optimal functional recovery. It has been observed that partially transected nerves often recover faster and more completely than those requiring surgical repair.[188] As the vast majority of nerve injuries are in continuity or partial transections, surgical decisions are usually postponed for 8 to 12 weeks.[192,193] Such a hiatus allows for more definitive assessment of severity, resolution of neuropraxia, and potentially, the maturation of the distal nerve stump.[7] Postponement of conservative therapy during this period, however, may lead to secondary injury, which will hamper subsequent functional return. Sharp lacerations are commonly explored within 72 hours as early repair in those situations leads to relatively good outcomes.[192] Yet, even when sharp laceration is the known causative agent, only 15% to 20% are observed to be complete at exploration.[193] Thus, in the vast majority of cases, conservative therapy is the first line of treatment and the cornerstone for any further intervention.

The extent of functional recovery hinges on the extent of injury. Surprisingly, a small number of residual motor units can lead to near normal motor function. Edds' study[47] of reinnervation in partially denervated muscles suggested that reinnervation and axonal sprouting could lead to useful function when 50% to 75% of axons were lost. No functional recovery occurred when greater than 80% of motor units were lost.[9] This finding is consistent with the poor prognosis seen in patients with acute inflammatory demyelinating polyneuropathy, whose electrodiagnostic evaluations reveal CMAP amplitudes less than 20% of the lower limit of normal.[194]

Rehabilitative measures do not improve the rate of reinnervation or reverse the end organ changes occurring specifically from denervation. They are indispensable, however, in the protection of the affected limb from secondary injuries derived from contractures, disuse weakness, edema, pain, and poor positioning. Regardless of whether surgical intervention is warranted early, after a delay, or not at all, these same principles are also employed to facilitate the attainment of maximal functional capacity in the face of residual sensory or motor impairment.

Maintenance of Range-of-Motion

Physiology

The upregulation of collagen synthesis following denervation portends the ominous development of myogenic contractures. Sunderland noted that the development of fibrosis and contractures is so prejudicial to restoration of function if and when reinnervation takes place, that it should be kept to a minimum by appropriate therapy.[9]

Increased flexibility results primarily from stretching the connective tissues within and around the muscle and tendon rather than the contractile elements of muscle. Connective tissues will progressively shorten when not opposed by a stretching force and will elongate when challenged with a constant stress.[195] Stretching, elastic or plastic, occurs when there is linear deformation of the fibers that leads to an increase in length. Elastic stretch occurs when elongation is produced with loading, followed by a recovery to resting length when the load is removed. Plastic stretch occurs when the elongation is maintained after removing the load.[196] Plastic elongation is the type necessary to improve flexibility of connective tissue shortened by edema, injury, or unopposed or imbalanced muscle tension. Four factors influence plastic deformation: (1)

amount of force, (2) type of force, (3) duration of force, and (4) tissue temperature.

Lehmann et al[197] documented that prolonged loading of a tendon leads to significantly greater increases in length than short term loading. This confirmed a more clinically based study by Kottke et al[195] who reported that short duration, high force stretching leads to high tensile resistance and little change in length, while prolonged, low force stretching leads to plastic elongation. Temperature increases to a therapeutic range of 40°C to 43°C decrease the viscosity properties of the connective tissue and maximize the effect of stretching.[197] In the Lehmann et al study,[197] stretching during heating led to significantly greater length increases than stretching after heating was completed.

Clinical Application

The efficacy of passive motion to prevent contractures in the face of peripheral nerve injury is well established (Figure 9-30). Pollock et al[198] brought affected joints through their full range passively for one set of 10 repetitions each day. The end range of each repetition was maintained for several seconds. No contractures developed in either the treated or control groups prior to 30 days postinjury. By 90 days, however, nearly 50% of the control group had developed contractures averaging 35°. Only 15% of the treated group developed contractures. The average severity of contractures in this subgroup was 20°. Institution of passive range-of-motion retarded the development of contractures, diminished their severity, and facilitated their resolution. Passive stretching has also been the principal method of averting contractures in neuromuscular disease. While the experience may not be perfectly applicable to isolated lower motor neuron injuries, the basic premise remains the same. Vigos[199] suggested that passive range-of-motion be initiated immediately and should be undertaken at least once or twice daily. Each session should include 10 to 15 repetitions with the end range held for a 10 to 15 count. Kottke[200] set similar parameters to be done twice a day. As strength improves, the patient should be encouraged to actively assist with range-of-motion. Once strength improves to better than antigravity, active range-of-motion should be continued by the patient even outside of formal therapy sessions.

The intensity of the stretch is crucial to the maintenance or improvement in range-of-motion. In situations where no inflammation is present and sensation is intact, muscles can be stretched vigorously. Stretching can be to just past the point of pain but the pain should abate rapidly with discontinuation of the stretch. Traumatic nerve injuries are commonly complicated lesions, coexisting with bone, vascular, and muscle injuries. Range-of-motion in these situations should be done only by highly trained therapists or physicians. Complications of overstretching, including hemorrhage, myositis ossificans, heterotopic ossification, and disruption of supporting structures, need to be obviated at any cost.[201]

Fig. 9-30. Passive range of motion of the phalangeal joints, administered to a patient sustaining radial nerve injury with weakness of the wrist and finger extensor muscles.

When stretching to correct connective tissue contractures in situations void of inflammation, edema, or hypesthesia, the elevation of tissue temperature should be accomplished through heating modalities. The choice of modality depends on the depth of the tissue to be heated. Deep muscles will require ultrasound diathermy. More superficial tissues such as the finger flexors will be adequately heated by superficial modalities such as moist heat. Concomitant, gentle, prolonged stretching should be applied across the joint once elevated temperatures are attained. The use of heat as an adjunct to stretching is relatively contraindicated in anesthetic regions; regions with impaired vascular supply; and in acutely injured, edematous areas.[201]

Strength Maintenance and Improvement

Physiology

Exercise therapy to preserve or enhance motor strength is integral to the comprehensive rehabilitation of persons sustaining peripheral nerve injuries. As previously discussed, over 60% of weakness experienced during such injuries can be attributed to disuse atrophy.[75] Incomplete reinnervation, whether from intrinsic factors such as muscle fibrosis or extrinsic factors including aberrant innervation by inappropriate axons or lack of reinnervation secondary to neuroma formation, may lead to residual weakness. Much of the understanding of the effects of strengthening partially denervated and reinnervating muscles is derived from experience during the poliomyelitis epidemics of this century. Related studies assessing the utility of strengthening exercise for neuromuscular disease may also be applicable. Herbison and colleagues[202–206] specifically tackled the questions of appropriate intensities, duration, and timing of strengthening exercises following peripheral nerve injury.

Clarifying Overwork Injury

The use of resistance training to improve muscle strength in neurologically intact individuals is unquestioned in the lay and medical literature. Its use in the management of peripheral nerve injury has not been as resolute. Concerns regarding provocation of declines in strength caused by overstressing immature neuromuscular connections or by over taxing a sparsity of competent motor units have been raised. Reports[207,208] of strength loss in individuals with predominantly lower motor neuron injuries undertaking strenuous activity have been largely anecdotal. Lovett[207] surveyed victims of the 1913 polio epidemic, commenting on three persons who presumably lost strength following prolonged overstressing of individual muscles. A highly cited source alleging overwork weakness by Bennett and Knowlton[208] commented on four cases of anterior poliomyelitis and one cervical spinal cord injury.

In contrast, numerous studies have confirmed significant improvements in strength in those who have sustained lower motor neuron injuries. The results of the study by Delorme and colleagues[209] clearly demonstrate that muscles weakened by poliomyelitis and normal muscles respond similarly to progressive resistance exercise. Exercise protocols comprised 2 exercises of 3 sets, 10 repetitions, with a 1-minute rest between sets, done 4 days per week. Similar results were obtained following a regimen of 20 maximum repetitions done at 1-minute intervals, 3 times per week for 5 to 10 weeks.[204] No muscles showed a permanent reduction in power. Of note, it was also observed that slight increases in power frequently accompanied great increases in work capacity.

While the exact pathophysiologic etiologies of postpolio syndrome have yet to be determined, overwork weakness has been touted as a possible cause. Even in this situation, many studies have established that monitored exercise protocols employing submaximal progressive resistance exercises can improve strength and do not precipitate untoward declines in strength.[210–213] A study by Agre and Rodriguez[214] showed that when rest periods were interspersed between isometric contractions of 40% isometric peak torque rather than doing the same amount of work constantly to fatigue, the ability to recover strength after activity was improved.

These polarized conclusions may reflect differences in the classification of weakness. Overwork weakness is defined as a prolonged decrease in both absolute strength and endurance of a muscle subsequent to a period of work. Impairment must be longstanding.[208] Because most of the literature supporting the verity of overwork weakness are case reports and studies of not significant longitude, it is difficult to judge whether they meet the definitional criteria. In several cases in which both upper and lower motor neuron injury was evident, alternative causes for a loss of strength may have also been at play. Lastly, these studies did not show histologic evidence of injury such as central nuclei, fiber splitting, or fiber degeneration.

The type and intensity of activity undertaken more than the activity itself may also explain the disparity. In those cases manifesting weakness after activity, individuals had engaged in prolonged, strenuous activities lasting hours at a time and were for the most part unsupervised. In the prospective studies, episodic bursts of high resistance maximal or submaximal exercise were performed for short periods each day. Evidence supporting this impression can be found in a series of experiments by Herbison and colleagues.[202–206] Wistar rats were subjected to sciatic nerve crush injuries and either soleus or plantaris tenotomies. Exercise was started at 2 weeks postinjury in one group and 3 weeks postinjury in the other group. The findings revealed increases in muscle weight and protein only in the group postponed 3 weeks. A mild decrement was observed in the group starting only 2 weeks postinjury. Thus, the group participating in the more intense exercise program developed a loss of muscle protein and weight. It should be understood that the exercise undertaken by these animals was prolonged and continuous. Also, all rats were autopsied at 4 weeks postinjury. It is unclear as to whether the changes seen were transient or persistent. It was concluded that a critical relationship existed between the number of contractile units and the intensity of exercise. When the intensity becomes too great for the fibers available, normal physiologic processes may become pathologic.[204]

A follow-up study[205] looked at a less intense form of exercise, swimming. In this situation, denervated rat muscles showed progressive increases in muscle weight and protein concentrations, directly proportional to exercise time and duration. As with most therapeutic applications in medicine, the prescription of strengthening exercise for peripheral nerve injuries must fall within its therapeutic range. The muscle must be overloaded to the extent warranted to overcome disuse atrophy and eventually to improve strength. The stress must also be below levels which may provoke injury.

Clinical Application

The determination of the therapeutic range for strengthening exercise varies directly with the current strength of the individual and the motor units involved. Commonly, electromyographic evidence of reinnervation precludes the onset of clinically observable muscle contraction. In these situations, surface EMG biofeedback might be employed to provide auditory and visual cueing to enable controlled firing of the motor units. Parameters based on the amplitude of the evoked potentials may be used to set goals regarding intensity and duration.[215–217]

Patients with incomplete injuries or those in the process of reinnervation may exhibit strength at all ends of the spectrum. When weakness precludes the voluntary movement of the affected joint through the full range-of-motion against gravity (ie, trace (1/5) or poor (2/5) strength), isometric strengthening exercises are most appropriate. An isometric contraction is a muscle contraction without movement across the joint. Peak isometric strength is the force that can be exerted against an immovable object.[218] In cases of trace and poor strength, the weight of the limb in the face of gravity provides adequate resistance.

While most of the literature regarding strengthening focuses on techniques to attain maximal improvement, Muller[219] assessed the basic requirements to maintain or modestly improve strength in skeletal muscle. All therapeutic prescriptions are founded on four variables that can be manipulated: (1) mode, (2) intensity, (3) frequency, and (4) duration. Mode is the type of exercise to be undertaken. Intensity is the percentage of the individual's maximum capacity. Frequency refers to the number of sessions over a period of time, and duration determines the length of each individual session.

Strength Maintenance

In the early stages following peripheral nerve injury, efforts should be made to maintain strength and overcome disuse atrophy. Commonly, these patients have sustained multiple trauma. Surgical procedures may be impending or the patient may still be in the intensive care unit. Maintenance of strength even at this juncture is vitally important as it will facilitate the rehabilitation process in the future. Surprisingly, very small amounts of exercise are needed to maintain strength. Muller[219] observed that 1 maximal isometric contraction, held for 1 second, performed once a week led to minimal but significant strength improvements. Spacing isometric contractions to 1 every 3 weeks caused moderate decrements from baseline strength. One maximum isometric contraction of 1-second duration done every other week generated mild increases in strength, which slowly returned to baseline by the 14th week.

Strength Improvement

At these modest levels of activity, increases in any variable (ie, intensity, frequency, or duration) will lead to increased strength enhancement. Goals may be attained more rapidly by employing a more rigorous program. Modulation of the duration of contraction has led to mixed results. Muller[219] observed no difference in strength outcomes when 1 isometric contraction was held for 1 or 6 seconds. Another study showed comparable strength gains when one maximal isometric contraction was held for 5 or 45 seconds. This paradox was explained by Mundale,[220] who showed that during 1 maximal isometric contraction, maximum tension could be sustained for no longer than 1 second. Yet, when variations in intensity and duration were assessed, differences in each variable affected outcomes. It was observed that one submaximal contraction, 65% of maximum of 1-second duration, performed once a day caused strength gains of 2.5% per week. One daily maximal contraction of 1-second duration improved strength by 3.33% per week. One daily maximal contraction of 6-seconds duration increased strength by 4% per week. Lastly, the greatest improvement of 5% per week was achieved by performing one set of 5 contractions, each 6 seconds in duration, spaced every 2 minutes. A total of 30 seconds of contractions were completed daily.[219] The minimal increase in strength gain observed when one 1-second and one 6-second maximal isometric contractions are compared may reflect additive gains during submaximal contraction. While maximal intensity can only be maintained for 1 second, less powerful contraction during the subsequent 5 seconds may still provide strength improvement.

Once antigravity strength has been achieved, isometric exercise may no longer suffice as the sole method of strengthening. A major drawback of isometric exercise is that strength gains are limited to the joint angle or muscle length at which the muscle is exercised. There is little transference of strength to dynamic activities.[221] It is reasonable to progress to exercises that will enable the production of strength increases throughout the entire range-of-motion. Isotonic exercises involve moving a constant load through a full range-of-motion with or without a changing velocity of movement (Figure 9-31). DeLorme and Watkins[222] are credited with establishing resistive exercises as rehabilitative tools to increase strength.

As previously mentioned, a specific application of DeLorme and Watkins'[222] method had been to improve strength of acute poliomyelitis victims. His program involved the use of progressive resistance exercises with increasing loads. Each individual determined the ten repetition maximum (TRM) for each muscle to be strengthened. The TRM is the greatest weight that can be lifted through the full range of motion 10 times only. Subjects would then perform 3 sets of 10 repetitions daily at 50%, 75%, and 100% of the TRM with 2 minutes of rest between sets. Each week a new TRM would be determined. The drawbacks to this exercise included difficulty in completing the final set of exercises due to fatigue and the fact that full motor unit recruitment was only accomplished during the last set.

The Oxford technique[223] reversed the DeLorme regimen by ordering the exercise sets with 100% TRM first, followed by 75%, and then 50%. With this regimen, fatigue caused by the 100% TRM set is offset by lower loads on the second and third sets.

Both of these techniques were based on the premise that only high weight, low repetition exercises produce strength gains. DeLateur and Lehmann[218] observed that strength gains may be obtained even at levels as low as 30% of maximum voluntary contraction. While there is little evidence to substantiate the provocation of overwork weakness by exercise of intensities described, it should be understood that submaximal exercise levels will improve strength.

Close evaluation of the patient is paramount. Herbison suggests monitoring strength on a daily

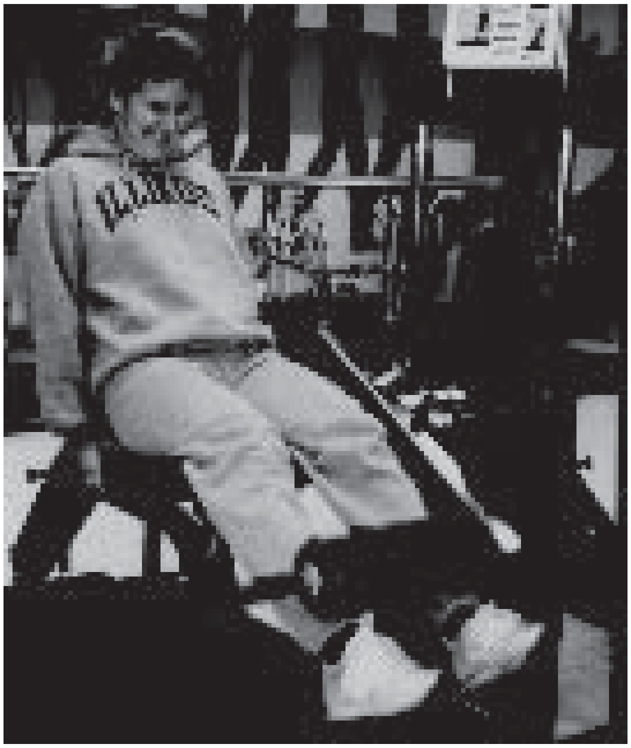


Fig. 9-31. Exercising knee extensors. Isotonic strengthening exercises utilize the movement of a constant load through a full range of motion.

basis (Gerald J. Herbison, M.D., Professor and Director of Research, Department of Rehabilitation Medicine, Thomas Jefferson University Hospital, Philadelphia, Pasylvania: Telephone conversation March 1993). Decreases in strength on the day following therapy or a digression in the ability to perform activities of daily living (ADL) should be an admonition that an excessive amount of exercise was attempted. Goals should be lowered and not progressed until they can be achieved without provoking unsatisfactory effects.

These exercise programs can also be used with isokinetic training. Isokinetic strengthening employs equipment that provides a set rate of velocity against which the person can exert maximum torque (Figure 9-32).[218] The velocity chosen becomes very important in isokinetic strengthening.[224] Training at slow velocities generates the greatest torque. Strength gains occur at the training velocity and can be seen when testing is done at even slower velocities. If strength testing is done at faster velocities than the training velocity, no evidence of increased strength is observed.[225]

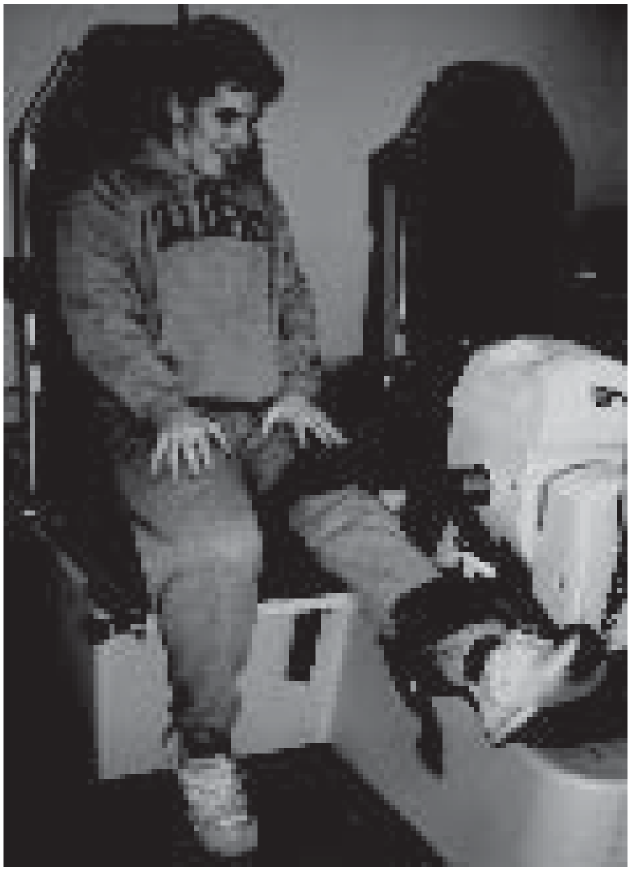


Fig. 9-32. Exercising knee extensors. Isokinetic strengthening requires the use of a machine, which limits the velocity at which a person can generate maximum torque.

A benefit to isokinetic training is that objective measures of torque production can be obtained that may reveal subtle persistent deficits. The biggest drawback is the reliance on special equipment which may not be readily available. There is no evidence that isokinetic strengthening is any more effective than isotonic training. Given their simplicity and universal availability, isotonic exercises are more commonly used.

Edema Control

Physiology

Efforts to ameliorate the development of edema must be expeditious. Traumatized limbs sustaining immobilizing nerve injuries are at great risk for the development of dense, restricting, connective tissue contractures. Edema facilitates the process of fibrosis in the affected region. Histological evidence of fibrosis may be seen as early as 4 days postinjury.[226] Extravascular fluid is also a ripe medium for bacterial growth and infection, which may seriously compromise any likelihood of subsequent functional recovery.

Clinical Application

Several methods to decrease edema, including range-of-motion exercises, elevation, external compression, and massage need to be integrated into an effective treatment plan.

Active range-of-motion. The maintenance of equilibrium between intravascular and interstitial fluid volumes is due in part to the contraction of skeletal muscle.[168] When possible, active motion performed by the patient through the full range-of-motion should be encouraged. Functional electrical stimulation may be a helpful adjunct in situations where strong muscle contractions cannot be performed because of neuropathic weakness (Figure 9-33).

Elevation. Utilization of gravity to decrease edema accumulation is also extremely helpful. Ideally, the affected limb should be kept above the level of the heart to ensure the best passive return of fluid to the central circulatory system. Great lengths should be taken to facilitate the most advantageous positioning throughout the day. When the patient is in bed, the limb should be supported by pillows or foam wedges. Distal portions of the extremities

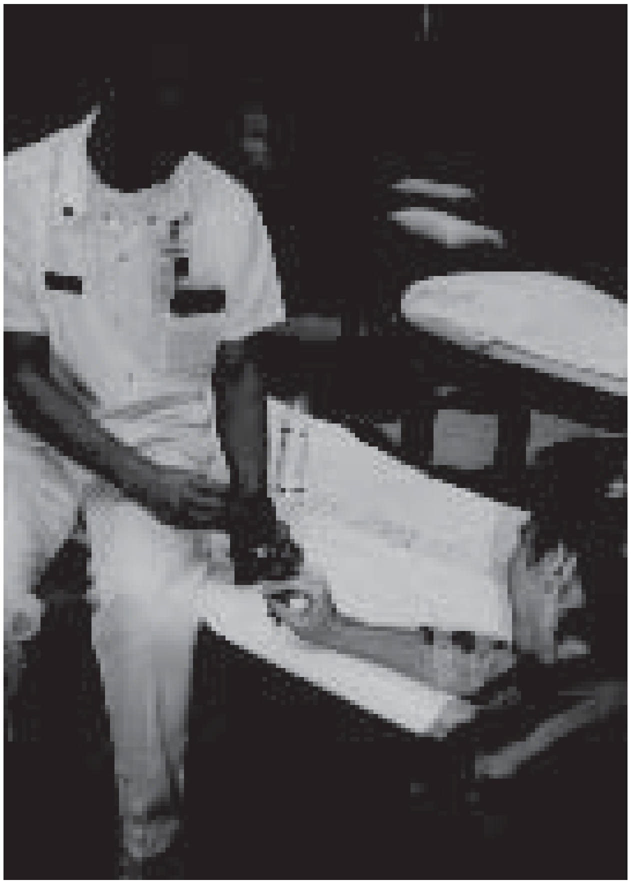


Fig. 9-33. Electrical muscular stimulation, applied to elicit muscle contraction and decrease the pooling of edematous fluid.

may warrant splinting in a functional position. At the wheelchair level, foam wedge laden arm troughs should be used to support the upper extremities. Affected lower extremities should also be elevated. Elevation above the level of the heart is difficult. At a minimum, the distal aspect of the limb should be kept higher than the proximal end. Dependent positioning should be avoided as much as possible.

External compression. External vascular support from gradient pressure elastic sleeves or stockings should be used continuously when edema is present. Isotonic compression gloves can also be used to combat swelling of the digits and dorsum of the hand.

Intermittent pneumatic compression may also be beneficial. The limb is placed in an inflatable sleeve. Pressure is applied intermittently. Pressures around 30 mm Hg can be applied for 5 to 6 hours in 3-minute cycles (ie, 2.5 min on, 30 s off). Alternatively, 60 mm Hg of pressure can be used for 30 to 60 minutes.[227] It is recommended that the maximum pressure applied should be less than the patient's diastolic blood pressure to prevent vascular occlusion and possible ischemic injury. During each session, the limb should be optimally elevated at a 45° angle. Gradient pressure garments should be applied immediately following treatment to help maintain gains made during mechanical compression.

Centripetal wrapping of the limb is another method commonly used to decrease the accumulation of interstitial fluid. Coban, a semiadhesive, elastic tape is applied distally, and circumferentially wrapped in a proximal direction (Figure 9-34). The limb is elevated for 5 minutes after application. Active range-of-motion is encouraged immediately following its removal. The treatment is repeated 3 times daily.

Massage. According to Knapp, the single best indication for the prescription of massage is reduction of swelling associated with trauma.[228] Effleurage (stroking) and petrissage (compression) methods are applied in a retrograde manner, mobilizing tissue fluids and assisting in proximal return to the intravascular circulation.

Orthotic Management

The rationale for application of orthoses to the nerve-injured limb are numerous. During the early phases of treatment, orthoses are used to limit motion, allowing for healing of the traumatized tissue. Proper positioning helps to prevent the development of contractures in unopposed muscles and overstretching of weakened muscles. Protection against joint and ligamentous injuries to insensate

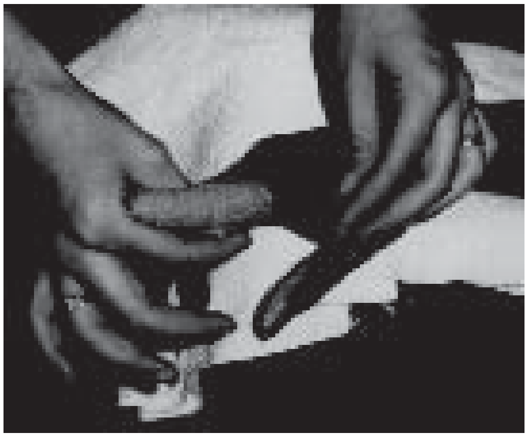


Fig. 9-34. Centripetal wrapping of the digits with Coban to decrease the accumulation of edema.

and weak limbs can be achieved through orthotic application. In the later phases, static and dynamic orthoses can be fabricated to enhance or substitute for lost function. The principles of orthotic management for musculoskeletal as well as nerve injuries are explored extensively in Chapter 11, Orthotics for the Injured Soldier, which is dedicated solely to the topic.

Hyperesthetic Desensitization

Hyperesthesia is a typical consequence of nerve injury. The instinctive perception of this abnormal sensory experience as injurious compels many patients to protect and not use the affected limb. The ominous consequences of disuse and immobility will further compound the loss of function and mounting anxiety if left untreated.

In 1976, a formal desensitization treatment program was developed at the Downey Hand Center.[229] Integration of psychological and physical principles provided an important foundation. The cognitive-behavioral psychological technique of systemic desensitization has been used in the treatment of emotional and anxiety disorders since the late 1950s.[230] Patients proceed through a hierarchy of anxiety-provoking situations. Each level is experienced repeatedly until the patient is comfortable with the situation. Step-wise progression continues until ultimately patients are able to cope effectively in the face of the circumstances they most fear. Hyperesthetic desensitization marries this concept with the overload principle, which contends that the body must be stressed beyond its current level of activity to adapt and enhance function.[231]

Clinical Application

Patients are exposed to increasingly irritating textures and vibrations through three 10-step modalities. The dowel modality incorporates textures ranging from moleskin to Velcro hooks, wrapped around the ends of ½-in. dowels, which are rubbed, rolled, and tapped on the hypesthetic region (Figure 9-35). Immersion into buckets of particles ranging from cotton to plastic squares is used in the particle modality (Figure 9-36). Exposure to increasing frequencies of vibration ranging from 23 to 100 Hz is used in the vibration modality (Figure 9-37, Table 9-9). Each modality is used three to four times per day. Progression is based on the patient's experience. Each day the patient chooses the level felt to be slightly irritating but tolerable for a 10-minute

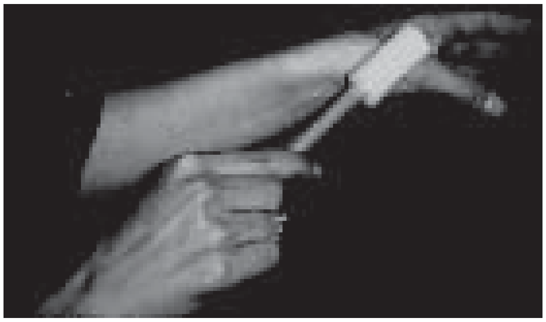


Fig. 9-35. Texture covered dowel, rolled, rubbed, and tapped on hypersensitive region.

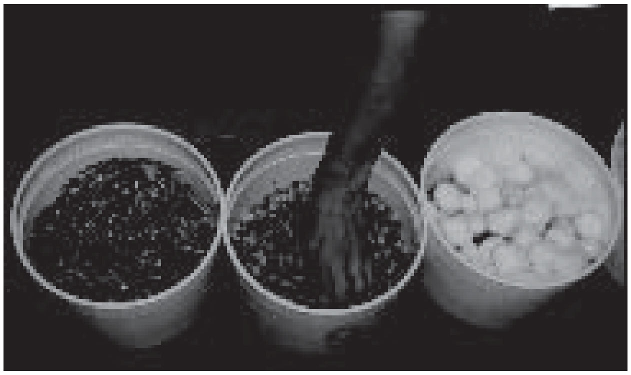


Fig. 9-36. Limb immersed in buckets of variably textured particles.

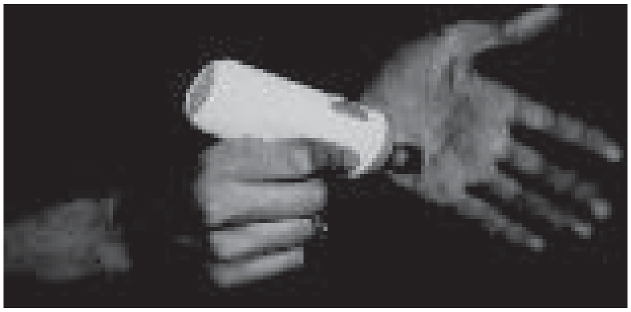


Fig. 9-37. Variable frequency vibrator providing stimulation to hypesthetic area.

TABLE 9-9

HIERARCHICAL STIMULATION FOR DESENSITIZATION THERAPY

Dowel Textures	Immersion Particles	Vibration (Hz)
Moleskin	Cotton	83 near area
Felt	Terrycloth	83 and 23 near area
Quickstick	Dry rice	83 near area, 23 intermittent on area
Velvet	Popcorn	83 and 23 intermittent on area
Semirough	Pinto beans Cloth	83 intermittent, 23 continuous
Velcro loops	Macaroni	83 continuous, 23 intermittent
Hard T-foam	Plastic wire Insulation pieces	100 intermittent, 53 intermittent
Burlap	Small BBs	100 intermittent, 53 continuous
Rug backing	Large BBs	100 continuous, 53 continuous
Velcro hooks	Plastic squares	Unlimited vibration

Source: Barber LM. Desensitization of the traumatized hand. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. Princeton, NJ: CV Mosby; 1984: 493-502.

period. In the Downey Hospital study,[229] progression to the next tolerable level usually occurred 2 weeks after initiation of treatment. The length of treatment averaged 7 weeks. Attainment of the highest level of tolerance of the dowel, particle, and vibration modalities was achieved respectively by 62%, 54%, and 22% of patients who had sustained crush injuries to the upper limb. Over 90%, however, noted that their perception of abnormal sensation no longer precluded their return to gainful employment.

The mechanism by which desensitization is achieved remains unclear. It has been suggested that mollifying fears of pain and disability is the most beneficial aspect of desensitization. Down regulation of sensitized nociceptors through stimulation of a different set of sensory fibers analogous to the gait theory, postulated by Melzack and Wall,[232] may provide a physiologic explanation. Neither of these hypotheses has been adequately explored in clinical trials.

Sensory Reeducation

The inherent difference between physical impairment and disability is a basic tenet on which much of rehabilitation medicine is based.[233] Frequently, great functional gains can be achieved despite minimal changes measured grossly on physical examination.

Physiology

The focus of sensory reeducation following nerve injury or repair is to retrain the patient to utilize what residual function remains to its fullest capability. The reserve capacity of sensory perception is not well established. It has been consistently observed, however, that a heightened sensitivity to certain modalities can be achieved as compensation for the loss of another. For example, normal two-point discrimination has been established as 3 to 5 mm.[234] Yet, those with impaired vision, who use tactile sensibility as their main mode of communication and interaction with the environment, may have two-point discrimination as fine as 1.5 mm.[235]

The success of sensory reeducation is founded on a sound understanding of which sensory modalities are essential for function and what level of intensity needs to be perceived. Moberg[236] noted that the presence of most sensory modalities tested on the standard neurologic examination do not correlate with function. Of the tests performed, perception of less than 12 mm on the Weber two-point discrimination test best predicted the ability to perform precision grip. Dellon[237] observed, however, that many persons with static two point discrimination greater than 12 mm functioned extremely well. Further experiments revealed that perception of moving two-point discrimination at the finger tips of less than 6 mm correlated with the ability to identify objects correctly. Sensory fibers which mediate the sensation of touch can be divided into two categories: slow-adapting and fast-adapting. Slow-adapting fibers transmit afferent volleys proportional to the intensity of pressure. They convey information regarding static, constant touch. Fast-adapting fibers respond not to levels of pressure but to abrupt differences and oscillations, experienced as dynamic or moving sensation. Thus, an integration between both static and dynamic pressure sensibility is needed for function.

Clinical Application

Sensory reeducation can be divided into two main categories: protective and discriminative reeducation.

Protective Sensory Reeducation

Patients with profound sensory loss are at great risk for inadvertently injuring the affected limb. Reeducation in this situation is compensatory and education of the patient is paramount. The patient gains an enhanced awareness of situations and stimuli which may place the limb in danger. Problems such as soft tissue injuries and Charcot joints, stemming from lack of proprioception and an inability to self-monitor motor function, and blisters and skin breakdown from overuse and abnormal sudomotor activity are brought to the patient's attention and are monitored scrupulously.[238]

Discriminative Sensory Reeducation

Wynn Parry[239] claims to have established the first formal sensory education program in 1966. Dellon and Jabaley[240] expanded on the concept and have developed a comprehensive approach based on specific levels of sensibility. Pain and temperature sensibilities are the first to return after nerve injury or repair. As discussed, aside from their important protective actions, they do not contribute to the functional capability of the limb. Low frequency (30 Hz) vibratory and moving-light-touch perception are next to return. Over time, constant touch and high frequency (256 Hz) vibratory perceptions return.

The early phase of discriminatory sensory reeducation centers on improving the ability to distinguish moving-light-touch from constant touch. This phase can begin only when low frequency vibration and moving-light-touch can be perceived; when resuturing has been performed, this is usually 4 to 6 months following surgery.[240] The exercises incorporate other sensory inputs to facilitate and reinforce greater awareness of the tactile sensation. Patients first observe visually the intensity, duration, and mode of touch. They then close their eyes and concentrate on the sensation itself. Lastly, they verbalize the sensory experience. Each session is brief, lasting only 10 to 15 minutes because of the great mental concentration needed to perform the exercises correctly. A minimum of one daily session is recommended but two or three are ideal.

Late phase discriminatory sensory reeducation encourages recovery of texture discrimination and object recognition (Figure 9-38). Commencement of the late phase follows return of the ability to differentiate constant and moving-light-touch or moving-light-touch discrimination of less than 6 mm. Fabrics, objects of variable size with rough and smooth edges, and household objects are manipulated in a similar format followed in the early phase. Visual reinforcement is used initially, followed by focused concentration on the tactile perception, and then verbalization.

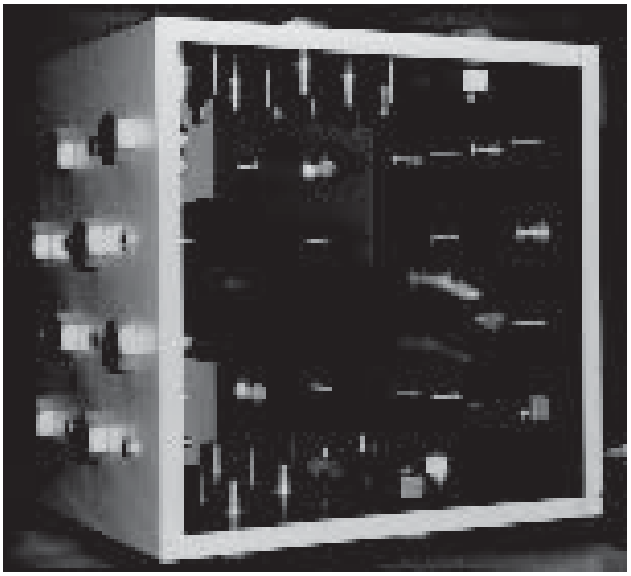


Fig. 9-38. The final phase of sensory reeducation involves sight obstructed manipulation and discrimination of common objects.

As discrimination improves, patients are advanced to activities such as picking objects from a bucket of sand, which requires the ability to discern an object from its background. Ultimately, ADLs with vision occluded are attempted.[238]

The benefits of the addition of sensory reeducation to surgical repair of nerve injuries to the upper extremities are encouraging and have become an integral part of hand therapy following nerve injuries. Several studies using Highet's grading system[241] (Table 9-10) have shown that over 80% of patients undergoing median or ulnar nerve repairs improved function to a grade of S3+ or better.[239,240] These findings strongly support the notion that functional return can be improved by repetitive training of sensory awareness.

Pain Management

Pain is a common perceptual sequela experienced by persons sustaining traumatic nerve injuries. Its

TABLE 9-10

CLASSIFICATION OF SENSORY RECOVERY

Grade	Level of Sensibility Function
S0	Absence of sensibility
S1	Recovery of deep pain sensibility
S1+	Recovery of superficial pain sensibility
S2	Recovery of superficial pain and minimal tactile sensibility
S2+	Same as S2 but with hypersensitivity
S3	Recovery of superficial pain and full tactile sensibility with resolution of hypersensitivity
S3+	Good stimulus localization but imperfect 2-point discrimination
S4	Full recovery of all sensibility

Source: Waylett-Rendall J. Sensibility evaluation and rehabilitation. *Ortho Clin North Am*. 1988;19:43–56.

aversive nature strongly deters interactions with noxious stimulation. The experience of pain during the aftermath of an injury can be extremely distressing to the patient. It is unusual for painful perceptions associated with nerve injuries to evolve into chronic, disabling experiences, per se.[242] They may, however, hamper attempts at rehabilitation and ultimately limit functional restitution. Aggressive early palliative treatment is an integral part of any comprehensive rehabilitation program.

Physiology

Asbury and Fields[243] divided neuropathic pain into two categories: dysesthetic pain and nerve trunk pain. Dysesthetic pain is associated with increased afferent input arising from hypersensitive, damaged, or regenerating nociceptive fibers. The sensation may be constant, intermittent, jabbing, or lancenating and is characterized as burning, tingling, crawling, or electric in nature. The distribution may be difficult for the patient to discern but usually falls within the cutaneous sensory distribution of the injured nerve. In neuromatous formations, the pain may be localized at the focus. Mechanical stimulation provokes discharge of the hypersensitive nerve endings.

Nerve trunk pain occurs in the face of more proximal injuries such as nerve root injuries and plexopathies. It is suggested that increased firing of nociceptive fibers within the sheaths of intact nerve trunks is the underlying cause. The sensation is described as a continuous deep aching and is occasionally noted to be knife-like or “like a toothache.” It may follow a dermatomal distribution but is more commonly noted as an ache involving muscles within the myotomal distribution.

A third type of pain may be experienced following complete transection of a nerve. In contrast to the two types of pain previously discussed, deafferentation pain is not dependent on damaged but rather on intact nerve fibers. It has been postulated that the loss of sensory input leads to abnormal somatotopic reorganization. Changes in the receptive fields at the spinal cord, thalamic, and cortical levels may lead to altered, painful perception as normal sensory input is relayed to inappropriate, reorganized regions of the central nervous system. Phantom limb pain is a specific type of deafferentation syndrome that follows amputation. A detailed discussion of the topic, including pathophysiology and potential treatment approaches, which are also applicable to deafferentation pain following complete nerve or nerve lesions, can be found in Chapter 4, Rehabilitation of the Lower Limb Amputee, in *Rehabilitation of the Injured Combatant*. Vol. 1, the first of the two Textbook of Military Medicine rehabilitation books.

Clinical Application

Early attempts at management of neuropathic pain were alarmingly unsuccessful. Maruta and colleagues[244] noted a success rate of only 20% during a 1-year follow-up of patients with chronic neuropathic pain. Current interventions that integrate modulation of both peripheral and central nociceptive transmission hold the promise of greater palliation of this complex and difficult problem.

Pharmacological Intervention

Antidepressants

To the authors’ knowledge, no studies have been conducted that confirm the efficacy of antidepressants in the treatment of traumatic, neuropathic pain. Their usage has been intuitive, based on research showing effective relief of pain caused by diabetic neuropathies and postherpetic neuralgia.[245–248]

The mechanism by which antidepressants provide analgesia is poorly understood. It has been hypothesized that chronic pain and depression commonly coexist.[249,250] Blockade of norepinephrine and serotonin re-uptake is postulated to compensate for relative deficiencies of these neurotransmitters. Improvement of the affective disorder is felt to enhance the ability to modulate and tolerate nociceptive inputs. An alternative theory suggests that pain reduction is an epiphenomenon of the sedation that is a common side effect of many antidepressant medications.[251]

A direct analgesic effect is supported by several clinical findings. The onset of pain relief usually occurs within the first 2 to 3 weeks,[245,246] while a large percentage of patients will not manifest improvement of depressive symptoms before four weeks of tricyclic antidepressant use.[252] The dosages needed to modulate pain are also lower than those needed for antidepressant effects. Several studies[245,248,249] have noted analgesic effect at doses ranging from 75 mg to 150 mg per day, while the recommended minimal dose for an adequate trial of a tricyclic antidepressant drug for depression is 150 mg per day and can be increased to as high as 300 mg.[252] Lastly, pain modulation has been confirmed in several placebo-controlled studies that also controlled for depressed patients.[245,248,253] A recent meta-analysis of 39 placebo controlled studies of antidepressant-derived analgesia in chronic nonmalignant pain concluded that the intrinsic analgesic properties and not the sedative or antidepressive effects of the drugs best explained the efficacy of this class of drug in pain modulation.[254] Several researchers have postulated that antidepressants may relieve pain through their ability to weakly bind to central opiate receptors[255]; however, the direct inhibitory action of enhanced levels of norepinephrine and serotonin in descending, pain-modulating pathways is more plausible. The relative importance of the individual neurotransmitters remains controversial. Several earlier studies suggest that those antidepressants that predominantly block serotonin re-uptake produce superior analgesic effects.[255] A more recent placebo-controlled study was undertaken comparing the effects of desipramine and amitriptyline (tricyclic antidepressants possessing both noradrenergic and serotonergic properties) with fluoxetine, a relatively pure serotonin re-uptake inhibitor. Intrinsic analgesic activity in the face of neuropathic pain was observed only in those drugs exhibiting central noradrenergic re-uptake inhibition. Pain relief was no better than placebo in the fluoxetine group. Max et al[245] postulate that norepinephrine is likely the primary metabolite responsible for pain relief, while serotonin is augmentative at best.

The choice of a specific antidepressant medication for the treatment of neuropathic pain is colored more by the side effect profiles of certain drugs than any proven difference in their effectiveness (Table 9-11). Placebo-controlled trials for amitriptyline,[245,247,248] desipramine,[246] nortriptyline,[256] and imipramine[257] have all shown good results in the relief of neuropathic pain. In certain situations, side effects such as sedation may be a corollary to the analgesia provided. Undesirable effects including insomnia, orthostatic hypertension, and anticholinergic effects (eg, dry mouth, constipation, urinary retention) are the most common reasons for choosing or switching to a certain drug. It should be stressed that these drugs are relatively contraindicated for use in persons with cardiac dysrhythmias, closed angle glaucoma, or urinary retention. Con-

TABLE 9-11

HETEROCYCLIC ANTIDEPRESSANT DRUGS USED FOR NEUROPATHIC PAIN

		SIDE EFFECTS			
Drug	Daily Dosage (mg)	Sedation, Hypotension	Orthostatic Effects	Anticholinergic Dysrythmic Effect	Cardiac
Amitriptyline	50–150	+++	++	+++	Yes
Desipramine	50–150	–	++	+	Yes
Imipramine	50–150	+	+++	++	Yes
Doxepin	50–150	++	++++	++	Yes
Nortriptyline	50–150	+	+	+	Yes
Trazadone	50–150	+++	++	0	Low

Source: Potter WZ, Rudorfer MV, Manji H. The pharmacologic treatment of depression. *N Engl J Med*. 1991;352:622–642.

sultation with an internist should be considered before administration in these instances.

It is recommended that treatment be started at a low dose. Many authors suggest an initial dose of 10 mg to 25 mg for tricyclic antidepressants.[248,253] The dose should be increased every 2 to 3 days, guided by the onset of untoward side effects. Ideally, a dose in the 75 mg to 150 mg range should be achieved. Lower doses have been observed to be beneficial in other pain disorders but, to date, only the higher doses have been used in studies documenting neuropathic pain relief.

Other antidepressant drugs including Doxepin and Trazodone have been shown to be effective in the treatment of chronic pain. No studies have been undertaken as yet to explore their utility in the treatment of peripheral neuropathic pain.

Neural Membrane Stablizers

Anticonvulsant drugs have been observed in noncontrolled trials to be effective in the treatment of shooting, stabbing pain but not background burning, aching pain.[258] They have also served as adjuncts to antidepressants in situations where neuralgic pain is refractory to antidepressant agents alone.[255] Swerdlow and Cundill[258] report rates of pain relief in posttraumatic nerve injury ranging from 64% to 92%. (Five anticonvulsants and one antiarrhythmic drug have been evaluated.)

Carbamazepine. Carbamazepine has been noted to be the most efficacious of the group and is chemically related to amitriptyline. It also causes the most problems in regard to side effects. Dizziness, unsteadiness, drowsiness, and gastrointestinal upset are commonly experienced during initial treatment. Many patients will develop a mild leukopenia, which should be monitored closely but does not necessitate cessation of the drug unless white blood cell levels fall below 3,500 cells/mm^3.[255] Rarely, but ominously, carbamazepine can induce aplastic anemia and agranulocytosis. Dosing should start low, 100 mg per day, and increase by 100 mg every 2 days with a goal of 600 mg per day in divided doses (ie, 200 mg three times daily [tid]). Administration can be increased to as high as 1,800 mg per day, although this is rarely tolerated or warranted. Close monitoring of all hematopoietic cell line levels and liver associated enzymes is essential.

Clonazepam. Clonazepam, a benzodiazepine, revealed similar effectiveness for neuropathic pain relief as carbamazepine but with a much better side effect profile.[258] Its major side effect is sedation, which may be potentiated by concomitant use of tricyclic antidepressants. Clonazepam use is relatively contraindicated in persons with significant renal insufficiency as its active metabolites are excreted by the kidneys. Persons with respiratory compromise may also be placed at risk secondary to enhancement of salivary secretions. The most significant drawback to its use, however, is the habituation and dependency which may develop in prone patients, as with any other benzodiazepine. Dosing should start at 0.5 mg per day and may be increased gradually, 0.5 to 1.0 mg every third day. An average therapeutic dose ranges from 2 to 10 mg per day divided into three doses.

Gabapentin. Recently a new anticonvulsant, gabapentin, has been shown to have potent analgesic properties, particularly in the treatment of neuropathic pain.[259–261] Gabapentin is structurally related to the neurotransmitter gamma-aminobutyric acid (GABA) but does not interact with GABA receptors and does not convert into GABA. In fact, the exact mechanism by which gabapentin produces analgesic effects remains unclear. The greatest advantage of the use of gabapentin for neuropathic pain is its remarkably low level of toxicity and side effects. The most common side effects are somnolence, dizziness, and ataxia. These are not common and were reported primarily when gabapentin was used adjunctively with other antiepileptic drugs. Dosing begins at 300 mg once daily and is increased by 300 mg every 2 to 3 days up to 300 mg tid. Given its relative safety and paucity of side effects, gabapentin has become the first choice among anticonvulsants for the treatment of neuropathic pain.

Phenytoin. Phenytoin is proposed to act at the level of the neurolemma Na$^+$ channels, enhancing membrane polarization and stabilization. A narrow therapeutic window limits its utility. Side effects commonly encountered include sedation, confusion, and ataxia. Ironically, a sensory polyneuropathy has been observed with chronic usage. Dosing usually begins at 100 mg twice a day and is increased by increments of 100 mg every 2 to 3 days to a daily level of 300 to 500 mg. A common schedule is 200 mg twice a day.[255] If symptoms have not improved by 3 weeks, the dose should be tapered off, as higher doses will only lead to toxicity.

Valproic acid. Valproic acid possesses similar analgesic effects as phenytoin.[258] Given its potential to induce profound, fulminant hepatic failure without warning, it is not commonly used for neuropathic pain relief, especially when more effective agents are available.

Mexiletine. It has been known for some time that intravenous administration of anesthetics such as lidocaine produce pain relief independent of their ability to block neuronal conduction.[262] Their usefulness in the control of persistent pain has been limited by the logistical difficulties of intravenous infusion of these relatively short-acting agents. The use of mexiletine, an oral lidocaine analogue has recently been reported in the effective management of diabetic neuropathic pain,[263–265] neuromatous pain,[266] and peripheral nerve injury associated pain.[267]

Mexiletine's analgesic properties stem from its ability to block Na^+ channels, stabilizing membrane ionic flow. Experimental studies in rats have revealed two possible sites of action. A peripheral effect was noted by Chabal et al.[266] Sensitivity to mechanical stimulation and spontaneous firing of injured axons was significantly reduced in sciatic neuromas. Woolf and Wiesenfeld-Hallin[268] suggest a central inhibitory effect at the level of the dorsal horn.

A graded response occurs with mexiletine administration, directly related to the dose. Most authors suggest starting at 150 mg per day and increasing by 150 mg every third day until 450 mg per day is achieved (150 mg tid). If satisfactory pain relief is not achieved, the dose can be increased by increments of 150 mg per day at weekly intervals to a maximum level of 750 mg per day (approximately 10 mg/kg/d) in divided doses.[265,267]

The most common side effects experienced by patients are gastrointestinal upset with nausea and occasionally vomiting. Dizziness, tremor, and a general perception of nervousness are also not uncommon. As with other antiarrhythmic agents, the potential for exacerbating underlying cardiac dysrhythmias is very real and potentially dangerous. Thus, all persons should undergo electrocardiographic evaluation prior to the commencement of, and periodically during, treatment with mexiletine. Its use is contraindicated in persons with second- or third-degree A-V block, not using a pacemaker. It is also strongly discouraged for treatment of pain in persons with ventricular dysrhythmias, congestive heart failure, or hypotension.

Substance P Inhibitors

Capsaicin. A naturally occurring alkaloid isolated from capsicum peppers, capsaicin was originally found to relieve the dysesthetic pain associated with herpes zoster,[269,270] and more recently, painful diabetic neuropathy.[271–274] No studies have assessed its potential use in traumatic nerve injuries. Capsaicin's analgesic action is derived from its ability to deplete substance P, a pain-modulating neurotransmitter from the terminals of nociceptive C-fibers. Initial exposure to capsaicin causes excitation of the thermal receptors, manifest as a burning sensation that lasts for 30 to 60 minutes. Repeated exposure, however, leads to the desensitization of these same fibers. Double-blind, vehicle-controlled studies evaluating not only pain relief but also functional capacities of persons with painful diabetic neuropathies revealed significant improvements along all parameters in the treatment groups.[272,274]

Several dosing schedules have been documented in the literature. Anecdotal improvement was seen using topical application of 0.25% capsaicin cream on the affected area tid.[271] Controlled trials have utilized a regimen of 0.75% capsaicin cream applied 4 times per day.[272–274]

As previously noted, the most common adverse effect of capsaicin use is a burning sensation that lasts for 30 to 60 minutes following application. In a study by Simone and Ochoa,[275] all subjects reported a decrease in the intensity and duration of discomfort over several weeks. Patients with severe hypesthesia, unfortunately, may find this early irritative effect intolerable, despite its self-limited course.

Concerns have been raised regarding the potential neurotoxic effects of long-term capsaicin use. High dose systemic administration in neonatal animals has caused irreversible destruction of nociceptive C-fibers.[276] Clinical doses of topically applied capsaicin, however, have not been observed to pose any threat to the integrity of sensory nerve fibers.[277] Apprehension about its use in sensory impaired individuals has also been debated. Capsaicin significantly raises the threshold for heat-pain detection. Thresholds for cold, touch, and mechanical stimulation are not affected.[275] Therefore, despite adequate preservation of protective sensation, patients with impaired baseline sensation should be counseled to take added precautions to prevent thermal exposure and injury. Alteration in heat detection is reversible and returns to baseline with cessation of capsaicin application.

Nonsteroidal Antiinflammatory Drugs

Nonsteroidal antiinflammatory drugs (NSAIDs) are the foundation of many pain management regimens. Their role in the treatment of neurogenic pain has been explored in only a paucity of studies. A single placebo-controlled experiment showed ibuprofen and sulindac to be more effective than placebo in the treatment of painful diabetic neuropathy.[278] Several anecdotal reports have also presumed benefit from their use.[279,280]

Following cell injury, mediators of vasodilatation and inflammation are released. Prostaglandins and leukotrienes, metabolites of arachidonic acid, are among the most potent and omnipresent inflammatory instigators. Prostaglandins produce pain by stimulating and sensitizing C-fiber nociceptors. C-fiber activity provokes further vasodilatation and release of inflammatory mediators. Thus, local injury leads to the cascading development of pain and inflammation at sites distant from the focus, termed neurogenic inflammation. Nonsteroidal antiinflammatory drugs' primary mode of action is the inhibition of prostaglandin production from their arachidonic acid antecedents. Nonsteroidal antiinflammatory drugs inhibit the activity of the enzyme cyclooxygenase, which catalyzes the addition of oxygen molecules to arachidonic acids to form prostaglandin precursors.[281]

There are currently over a dozen NSAIDs approved for use in the United States. Interestingly, response to any single agent is unpredictable. The class of agent also has little bearing on the success of pain moderation. The choice of a specific drug is based on side effect profiles, the potential for adverse effects, dosing schedules, and ultimately, cost. Excellent reviews of the pharmacologic properties and physiologic effects can be found in several texts, to which the reader is referred.[281,282]

In addition to the benefits gained from prostaglandin inhibition, many of the adverse effects of NSAIDs can also be ascribed to the impediment of their production. Gastritis and peptic ulceration are associated with the loss of mucosal protection afforded by prostaglandin-E_2. Nephrogenically produced prostaglandins act as endogenous angiotensin II inhibitors, blunting their vasoconstrictor and antidiuretic activity.[63] Patients with severe hypertension or congestive heart failure may be at risk of symptom exacerbation caused by NSAID prostaglandin inhibition. Platelet aggregation inhibition prolongs the bleeding times of patients taking most NSAIDs, with the exception of the nonacetylated salicylates. Nephrotoxicity and rarely hepatotoxicity have occurred with chronic usage. Nonsteroidal antiinflammatory drug use is also relatively contraindicated for treatment of patients with severe allergies or reactive airway disease. Prostaglandin inhibition shunts arachidonic acid precursors toward the 5-lipoxygenase pathway. Leukotrienes, the slow-reacting substances of anaphylaxis, are the ultimate product and may exacerbate symptoms.

Experimental Therapeutics

Experimental treatment with clonidine and calcium channel blockers have proved to be effective pain relievers in some cases, but in others there appears little relief was achieved.

Clonidine. Clonidine, an alpha$_2$-adrenergic agonist, has been reported to decrease pain associated with cancer,[283] arachnoiditis,[284] and herpes zoster.[285] Its precise action is unclear. Possible mechanisms include postsynaptic inhibition at the level of the spinal neurons, presynaptic modulation of nociceptive fiber activity, or inhibition of peripheral or central sympathetic activity.[286,287] A study[286] of the efficacy of transdermal clonidine (0.3 mg/d) for painful diabetic neuropathy failed to show a statistically significant effect. It was suggested by the authors, however, that reproducible pain relief was achieved in a subgroup and that the sample size and study design may have negatively impacted on the study outcomes.

Calcium channel blockers. Studies exploring the effects of calcium channel blockers on nociception are in their infancy. Kavaliers[288] noted that the administration of diltiazem or nifedipine augmented the endogenous production of enkephalins and raised pain thresholds. Calcium channel blockade has also been shown to potentiate the effects of exogenously administered opiates.[289] Gurdal and colleagues[290] showed potential antiinflammatory and nonopioid antinociceptive effects. Nicardipine was found to be highly effective, decreasing pain responses in rats by upward of 90%. Only partial results were obtained with verapamil and diltiazem. The role of cellular calcium ion flux has also been implicated in the actions of tricyclic antidepressants. Administration of nifedipine in conjunction with imipramine has been shown to enhance analgesic effects in Wistar rats.[291] Further controlled trials in human subjects are expected for these drugs and may expand the alternatives in the management of pain.

Electrical Stimulation Intervention

Transcutaneous electrical nerve stimulation (TENS) has been used to modulate pain for more than two decades. Despite controversies regarding mechanisms of action and efficacy, its use in the management of neuropathic pain has become nearly common. Few controlled studies have been performed examining its effectiveness in the face of peripheral nerve injuries. Rates of pain relief ranging from 50% to over 80% have been noted in uncontrolled trials.[292,293] Two methods of application are commonly used.

Conventional TENS

Conventional TENS employs high frequency (60 to 200 Hz) impulses of low intensity. Pulse widths range from 2 to 50 µs.[294,295] It is hypothesized that this afferent stimulation inhibits nociceptive inputs at the presynaptic level. Sjolund observed temporary diminution of transmission from nociceptive C-fibers in Wistar rats during peripheral application of TENS. The response was graded. Decreased firing was more pronounced in the 80 to 100 Hz range than at lower frequencies.[296]

The onset of analgesia is virtually immediate, following application of conventional TENS and rapidly abates when stimulation ceases. Of electrotherapeutic modalities available, this is undoubtedly the best tolerated and can be used continuously for hours at a time.

Acupuncture-like TENS

Low-frequency (0.5 to 10 Hz) stimulation at an intensity provoking muscle contraction is utilized in this form of TENS. Pulse widths range from several hundred to 1,000 µs.[294,295] In contrast to conventional TENS, acupuncture-like TENS analgesia has been linked to increased release of endorphins along descending central pain modulating pathways.[296] Acupuncture-like TENS is applied for 20 to 30 minutes. The onset of analgesia is delayed, occurring 30 minutes following completion of the stimulation. Pain relief may last for hours to days. The intensity of stimulation is poorly tolerated by many patients. Its use is frequently reserved for patients not responding to conventional TENS and those with predominantly deep aching pain.

Technique

Electrode placement and stimulation parameters continue to be as much art as science and herein lies the difficulty assessing the efficacy of this modality. Responsiveness to analogous applications may vary widely between individuals with similar pathologies. Electrodes can be placed in any number of configurations. A common method is to apply the electrodes on the periphery of the painful region with stimulation in a parallel or diagonal direction across the painful site. Electrodes can also be placed above or below the painful region. Stimulation along the proximal aspect of the nerve, in a noninjured region or even at the paravertebral levels, has been advocated (Figure 9-39). Pain relief has even been observed with placement of the electrodes on the contralateral limb.[294] Rarely is direct application on the painful region helpful or tolerated by the patient. Once electrodes are secured, the patient using conventional TENS is instructed to increase the intensity until a subtle tingling sensation is perceived. An inability to decrease pain perceptions by 50% or more within 30 to 60 minutes warrants repositioning of the electrodes and a trial at the new position.[295]

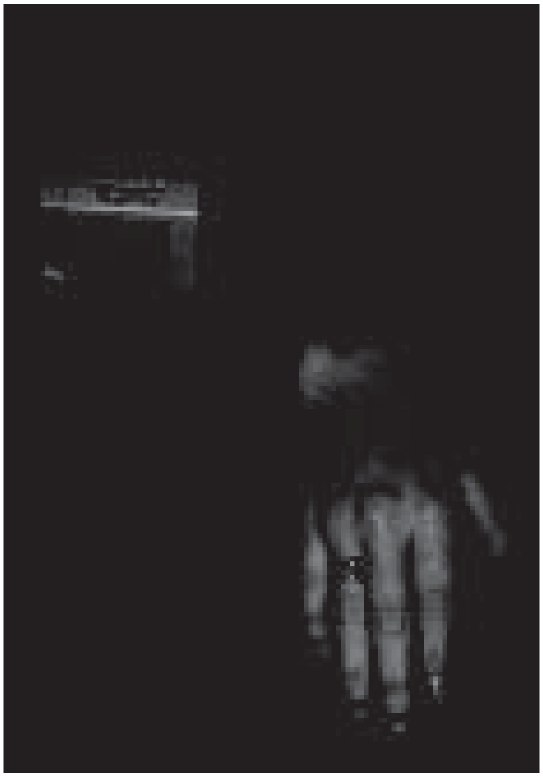


Fig. 9-39. Conventional transcutaneous electrical nerve stimulation (TENS) applied above the level of injury, on the edges of the sensory distribution.

Complications

Medical problems arising from the use of TENS are exceedingly rare. Skin irritation from the electric current, the tape used to secure electrodes, or the electrodes themselves are the most common complaint. These can be easily obviated through the use of self-adherent, disposable electrodes and repositioning the electrodes during subsequent applications. The use of TENS is contraindicated in patients with demand-type pacemakers. The TENS pulses may be misinterpreted by the pacemaker as heart beats and may inadvertently inhibit or overdrive the pacemaker.[297] It is also suggested that electrodes not be placed on the anterolateral region of the neck to avoid hypotension and bradycardia from stimulation of the carotid body and vagus nerve.

Electrical Muscle Stimulation

Enhancing regeneration of injured nerve fibers and reversing the effects of denervation atrophy have been the rationale for stimulating denervated muscle for nearly a century. There is little consensus in the literature, however, supporting its efficacy,[298] and in fact, several studies[43,299] suggest that it may ultimately impede the regenerative process. Much of the confusion stems from the disparate methods used to study its utility. Variables including the duration, intensity, frequency, and mode of application are far from uniform, differing in practically every study. Subjects, too, range from small amphibians to humans. Unfortunately, size and species do not account for the variability as polarized conclusions have been drawn within species groups.

Aside from the lack of conclusive evidence corroborating its effectiveness, a more basic question needs to be raised. Even if we accept the biased premise that electrical stimulation does retard atrophy and enhances nerve regeneration, are these benefits clinically relevant? If methods are extrapolated from animal studies, a substantial amount of therapy time must be dedicated to the administration of electrical stimulation. Time and labor focused on preventing secondary complications and improving motor and sensory function will be sacrificed. Stimulation levels producing tetanic contractions have been necessary in those studies reporting good outcomes. The loss of innervation requires the application of strong currents to achieve this effect. It is unlikely that many patients will be able to comply with such a regimen for any length of time.

The benefits of electrical stimulation of denervated muscle are equivocal at best. Compelling evidence clearly establishing its usefulness has yet to be presented. Its utility as a component of the rehabilitative treatment armamentarium can not be justified at this time.[9,294,298]

CAUSALGIA (COMPLEX REGIONAL PAIN SYNDROME, TYPE-II)

A unique pain syndrome associated with peripheral nerve injuries was first described in 1864 by Mitchell, Morehouse, and Keen,[300] physicians for the Union Army during the Civil War. In their monograph *Gunshot Wounds and Other Injuries of Nerves*, they established the first description of causalgia:

> It is a form of suffering as yet undescribed and so frequent and terrible as to demand from us the fullest description. In our early experience of nerve wounds, we met a small number of men who were suffering from a pain which they described as "burning" or "mustard hot" or as "a red hot file rasping the skin." In all of these patients and in many later cases, this pain was an associate of the glossy skin.… The part itself is not alone subject to an intense burning sensation, but becomes exquisitely hyperaesthetic, so that a touch or a tap of the finger increases the pain. Exposure to air is avoided by the patient with a care which seems absurd and most of bad cases kept the hand constantly wet, finding relief in the moisture rather than in the coolness of application.[300]

Causalgia (literally: burning pain) specifically applies to a symptom complex of burning pain, hypesthesia, vasomotor instability, and dystrophic changes occurring after injury to a major peripheral nerve or plexus.[301] The terms causalgia and reflex sympathetic dystrophy (RSD) have been used interchangeably in the literature. Recently, the International Association for the Study of Pain (IASP) has developed a revised taxonomy for these disorders. The overall term, Complex Regional Pain Syndrome (CRPS), requires the symptom complex described above for causalgia to be applicable. CRPS-Type I corresponds to RSD and occurs without a definable nerve lesion. CRPS-Type II corresponds to causalgia and requires the presence of a definable nerve injury.[302] The term sympathetically maintained pain (SMP) describes the presentation of continuous burning pain with mechanical allodynia, following a history of physical trauma in the painful area that is relieved by sympathetic blockade.[303] Sympathetically maintained pain can occur in association with either CRPS-Type I or Type II.[302]

The incidence of CRPS-Type II in nerve-related injuries has varied during different conflicts. A rate as high as 32% was originally reported during the Civil War.[300] More recent statistical revisions have lowered that rate to closer to 15%. Mayfield[304] reported that 4% of nerve-injured soldiers developed CRPS-Type II during World War II. Kirklin et al[305] noted an incidence of 2% during the same conflict, while series collected independently by Nathan[306] and Sunderland and Kelly[307] documented incidences of 14% and 12%, respectively. Rothberg et al[308] observed an incidence of 1.5% during the Vietnam War from 1964 to 1973. In two recent Middle East conflicts, rates closer to those observed during World War II have been documented. The assessment by Jebara and Saade[309] of nerve injuries sustained from 1975 to 1985 during the Lebanese Civil War revealed an incidence of almost 6%. Dillingham et al[3] documented 8% of all nerve injured soldiers evaluated by U.S. Army Physical Medicine and Rehabilitation Services as manifesting symptoms of CRPS-Type II during the Persian Gulf War.

While CRPS-Type II may occur after injury to any nerve, those nerves carrying the lion's share of sympathetic fibers are more commonly associated. In the upper extremity, injuries to the brachial plexus or median nerve are overwhelmingly involved. In the lower extremity, strong associations with sciatic or tibial nerve injuries have been established. Injuries to these nerve trunks accompany 83% of all cases of CRPS-Type II.[310]

Cardinal Characteristics

The vague definition of CRPS has led researchers to further clarify the salient features of this symptom complex. Four cardinal characteristics have been identified: (1) pain, (2) sensory abnormalities, (3) autonomic instability, and (4) trophic changes.

Pain

The pain associated with causalgia is undoubtedly the most disabling component. Classically, it is described as an intense burning sensation. It is nondermatomal in distribution and usually involves the distal aspect of the limb. Cases have been reported in which the pain spreads proximally and even to other limbs.[311] The onset of this type of pain quickly follows the infliction of the inciting wound. A compilation of series from World Wars I and II noted 89% of cases manifest symptoms within the first week following injury.[310] With time, the pain may change to a deep gnawing, aching experience. The pain may be at any intensity but commonly is severe enough to disrupt sleep and encumber the affected limb.

Sensory Abnormalities

Aberrant sensory perceptions are commonly experienced by patients. *Hyperalgesia* describes a lowered threshold to painful stimulation. Noxious stimuli that might normally be perceived as merely uncomfortable are felt to be excruciatingly severe. Another altered sensibility commonly experienced, termed *allodynia*, is the perception of pain from innocuous stimulation such as mechanical or thermal stimuli. Not uncommonly, patients describe unbearable pain from movement of the fine hairs on the limb by the sheets on the bed or a breeze blowing through the room. *Hyperpathia* may also be experienced. In this situation, there is an increasing sensitivity to repetitive stimulation. For example, a finger tap may not be painful initially, but as it is repeated, tolerance decreases and the once tolerable stimulation becomes less bearable. This amplification can be explosive. There is commonly after-sensation and radiation of the discomfort.[312]

Autonomic Instability

Alterations in the function of tissues innervated by the sympathetic nervous system help to differentiate CRPS-Type II from more common neuropathic pain syndromes. Sympathetic cell bodies are located in the intermediolateral gray matter of the spinal cord, extending from the eighth cervical level to the second lumbar level. The myelinated preganglionic axons synapse at the paravertebral ganglia. These white rami may traverse several levels of ganglia before synapsing. Acetylcholine is released at the terminals of all autonomic preganglionic fibers. The postganglionic axons are unmyelinated C-fibers that run with the anterior spinal motor nerves. The majority of postsynaptic fibers release the neurotransmitter norepinephrine. However, sudomotor fibers controlling sweat glands release acetylcholine.[313] Decreased sympathetic activity may be observed very early on, manifest by vasodilatory changes of warmth, erythema, and edema. Hypohidrosis is also observed as vasomotor and sudomotor dysfunction usually coexist. After several weeks to months, sympathetic hyperactivity may also occur. Cool, pale, cyanotic, sweaty limbs, reflecting vasoconstriction and hyperhidrosis, may be discerned.

Trophic Changes

Changes in the integument occur early. Gradual thickening and coarsening of the skin and the development of pitting edema are observed within days to weeks of the initial injury. Hair and nail growth is initially accelerated. In later stages, the skin thins and becomes taut and shiny. Nails become brittle, thickened, and pitted. Ultimately, hair and nails may fall out.

Muscles, ligaments, and tendons shorten and begin to develop contractures. The synergistic effects of immobility due to pain and the presence of edema spur this devastating evolution. As contractures progress and muscles weaken from disuse, joint stiffening and ankylosis may occur.

Bony changes are also seen incipiently and can be seen on bone scan within the first 2 weeks. Patchy, periarticular osteoporosis appears, usually at the metacarpophalangeal (MCP) or metatarsophalangeal (MTP) joints within weeks. Increased vascular flow, possibly related to abnormal sympathetic activity, leads to more generalized involvement. With time, it may become difficult to differentiate diffuse osteoporosis associated with causalgia from that merely secondary to immobility related to pain.

Clinical Staging

DeTakats'[314] descriptive analysis of five cases of RSD (CRPS-TypeI) in 1937 laid the groundwork for further codification of the course of the syndrome. Bonica[315] later grouped the signs and symptoms of untreated cases into three chronological stages. The utility of such staging should be viewed cautiously. Commonly, different signs progress at variable rates. It may become difficult to absolutely classify the severity of the syndrome. The evolution of CRPS-Type II runs along a spectrum, and therefore, the staging system is at best a gestalt view of syndrome severity. Dogmatic application of the staging paradigm may lead to the exclusion of true cases.

First Stage (Acute)

Within days to weeks of the inciting injury, the constellation of pain perceptions and abnormal sensibilities previously described begin. The pain is amplified by emotional stressors, auditory and visual stimulation, and especially by movement of the affected limb. A sodden, pitting edema develops distally. The skin is red, warm, and dry; hair and nail growth is accelerated. During the very early part of the first stage, no abnormalities may be seen on roentgenography. Later, the onset of periarticular osteoporosis is seen. Increased uptake on all three phases of technetium 99m bone scanning is seen in the periarticular regions of the small joints of the affected limb. The acute stage may last for days and resolve spontaneously. Alternatively, it may be recalcitrant, persisting for upward of 6 months.[316]

Second Stage (Dystrophic)

Between 3 and 6 months after onset, the signs and symptoms may evolve into the dystrophic stage. Pain and abnormal sensibility remain similar to that seen during the first stage. The integument becomes cool, pale, and cyanotic. Hair growth wanes and nails become thickened, brittle, and ridged. Edema takes on a firm, nonpitting consistency. Muscle atrophy begins as do contractures of all the soft connective tissues. The spotty osteoporosis seen during the later phase of the first stage becomes more generalized, commonly affecting the epiphyseal regions of the bones. Technetium 99m bone scans reveal normalization of the blood velocity and pool phases with persistently increased uptake during delayed fixation. The second stage can last for many months.[316]

Third Stage (Atrophic)

During the last stage, severe, atrophic changes resistant to treatment develop. Pain and sensory abnormalities may remain steady or rarely decrease in intensity. The skin becomes cool, smooth, glossy, and pale. The principal change however, is atrophy. Marked wasting of muscle and subcutaneous fat occurs, leading to taut stretching of the skin superficially over the osteoporotic bones. Joint contractures progress, culminating in cartilage destruction, joint subluxation, and eventually, ankylosis. Roentgenograms show severe, diffuse osteoporosis, while bone scans reveal decreased uptake during the flow and pool phases with normalization of the later fixation phase.[316]

Pathophysiology

The multitude of theories regarding the etiology of CRPS decries the lack of firm understanding of its pathophysiologic mechanism. The profound diminution of pain following sympathectomy, noted as early as World War I by Leriche (cited in Loh and Nathan),[317] has implicated the sympathetic nervous system as in some way involved in the perpetuation of this pain syndrome.[318] Several heuristic theories have recently been postulated to explain the inciting causes and alterations in the peripheral and central nervous systems that may perpetuate the syndrome even after resolution of the initial injury.

Specific types of sensory inputs are transmitted along particular classes of nerve fibers. Painful, injurious stimulation is conducted afferently along myelinated A-delta fibers and unmyelinated C-fibers. The ability to respond to several types of noxious stimuli or exclusively to a single form of nociceptive input varies between fiber types. Moderately intense mechanical stimulation is transduced through A-delta high threshold mechanoreceptors (HTM). A-beta myelinated mechanothermal nociceptors (MMTN) respond to heat greater than 45ºC and to intense mechanical stimulation. Both the A-delta HTM and MMTN fibers terminate along several dorsal horn laminae, predominantly laminae I, IIo, V, and X. C-polymodal nociceptors (C-PMN) are most sensitive to strong prolonged stimulation by temperatures ranging from 45ºC to 51ºC, intense mechanical stimulation or chemical irritation. C-PMNs terminate at laminae I, IIo, and V. A-beta low threshold mechanoreceptors (LTM) are nonnociceptive, large myelinated sensory afferents. These fibers normally transmit light touch and subtle mechanical stimulation. A-beta fibers terminate at laminae II through V. Some penetrate into the ventral horn, directly synapsing with motor neurons, while others ascend in the dorsal columns.

Of the dorsal laminae mentioned, lamina V is suggested to be integral in the establishment of sympathetically maintained pain syndromes. The most common cells found in the lamina are wide dynamic range (WDR) or multireceptive neurons. These nerve cells will respond to stimulation from not only A-delta and C-PMN but from A-beta LTM fibers. It has been suggested[316] that these WDR neurons may be sensitive to spinal and supraspinal modulation. While much research has focused on the roles played by individual components of the nervous system in the maintenance of sympathetically maintained pain, it is ostensible that peripheral and central alterations are inextricably related and likely confer perpetual changes upon each other.

Central Changes

Roberts and Foglesong[319] hypothesized that sensitization of WDR neurons in the dorsal horn is the pivotal occurrence driving sympathetically maintained pain syndromes. Trauma to a peripheral nerve, in the case of CRPS-Type II, causes nociceptive transmission along unmyelinated C-PMN fibers to the laminae of the dorsal horn, including WDR neurons in lamina V. Kenshalo et al[320] suggested that repeated volleys of C-fiber stimuli increase the sensitivity of WDR neurons to subsequent stimulation. As WDR neurons receive afferent volleys not only from peripheral nociceptors, nonpainful stimulation such as light touch transmitted by A-beta fibers also triggers amplified responses in the primed spinal neurons.

The WDR neuronal response furnishes an important component of ascending nociceptive information. Under normal circumstances, the low frequency and intensity of A-beta stimulation is unlikely to trigger the large bursts of activity in the WDR neurons that are perceived supraspinally as pain. Sensitized WDR cells, however, may produce exaggerated outputs in the face of normal A-beta stimulation. Allodynia and hyperpathia are manifestations of this aberrant, heightened sensitivity to nonnoxious stimulation.

Peripheral Changes

The suggestion that nonnoxious stimulation might be integrally involved in the development of sympathetically maintained pain was put forth initially by Loh and Nathan.[317] Their presumption was further substantiated by Campbell et al.[321] Differential nerve blocks were performed on patients with long-standing nerve-injury–associated hyperalgesia. Selective blockade of large myelinated A-beta fibers led to ablation of the abnormal sensation, corresponding to the loss of light touch sensibility. Sensory input transmitted by C-PMN and A-delta fibers remained intact. Selective blockade of C-PMN and A-delta fibers did not alter the perception of hyperalgesia. The unique susceptibility of A-beta fibers to sympathetic stimulation, as observed in several studies, corroborates their participation in sympathetically maintained pain syndromes.

Sympathetic Nervous System

The precise changes occurring centrally and peripherally, while better understood, continue to be debated. The implication of the sympathetic nervous system, however, has been suspected for close to a century and has rarely been contested. Stimulation of sympathetic ganglia at frequencies as low as 5 Hz have been observed to activate specific subsets of dorsal horn spinal neurons. Sympathetic stimulation failed to trigger neurons receiving input solely from A-delta high threshold neurons. Nearly 50% of WDR spinal neurons were activated.[322] Of the WDR neurons stimulated, studies have observed anywhere from 15% to 56% were composed of those receiving input from A-beta LTM.[320,323] Roberts et al[323] contend that increased sympathetic output is not necessary to drive the pain cycle. Normal sympathetic tone, as simulated by low-frequency stimulation causes firing of A-beta fibers in the periphery. This is in line with Torebjork's conclusion that there is little evidence from microneurographic recordings to support the contention that sympathetic outflow is increased in sympathetically maintained pain syndromes.[324] In the face of sensitized WDR spinal neurons, normal firing of sympathetically stimulated A fibers would be perceived as painful stimuli.

Roberts[303] has elegantly constructed a paradigm in which central neuronal changes, initiated by a peripheral injury, might be maintained by normal sympathetic activity, even after the original painful stimulation has resolved. It also provokes a number of questions:

1. If anywhere from 54% to 85% of WDR neurons are not triggered by sympathetically stimulated A fibers, are there other peripheral fibers that might be stimulated adrenergically?
2. Are there other peripheral mechanisms that might stimulate sensory fibers, maintaining WDR neuronal sensitization and triggering abnormal pain experiences?

Peripheral C-Polymodal Nociceptors

The strongest evidence for the additional involvement of C-PMN fibers is derived from studies of CRPS-Type II, in which the symptom complex is induced by peripheral nerve injury. The works of Devor and Janig[325] and Wall and Gutnik[326] have shown that afferent nerve fibers entangled in neuromas do respond to sympathetic stimulation as well as to local application of norepinephrine. Sato and Perl[327] observed that normal C-fibers did not respond to adrenergic stimulation. Partial injury of mixed nerves, however, led to situations in which C-PMN fibers developed enhanced sensitivity to both sympathetic stimulation and peripheral exposure of norepinephrine. Sensitization of C-PMN fibers to adrenergic stimulation started as early as 4 days after the inciting injury and peaked during the second week. This time frame mirrors the onset of sensory abnormalities and pain observed in clinical settings of CRPS-Type II.[310] Importantly, sensitization to adrenergic stimulation was not focused at the site of injury. It was noted to be most profound at the nerve endings. While these findings may have important implications in CRPS-Type II, it is unclear whether they can be generalized to nonnerve-injury-associated sympathetically maintained pain syndromes.

The role of peripheral instigators in the development and maintenance of CRPS-Type II cannot be underestimated. Undoubtedly, the overemphasis on ablation of only sympathetic efferents has led to many treatment failures. Following local injury and stimulation of C-PMN fibers, there is commonly the development of edema, caused by a reflex disturbance of vasoconstrictor outflow.[328] As the edema spreads, C-PMN fibers not initially in the region of injury are stimulated, causing a perpetuation of neurogenic inflammation. Less peripheral stimulation is needed to provoke firing of the C nociceptors once they have become sensitized. Thus, an escalation of C-PMN firing ensues, maintaining neurogenic inflammation, causing further sensitization of the C-fibers, and ultimately, sensitizing and perpetuating the firing of WDR spinal neurons. The development of edema, coupled with immobility induced by pain, spawns a new problem: soft tissue contractures. Movement of contracted joints excites muscle and joint nociceptors, adding to the sensitizing volleys converging on the WDR neurons.

Summary

Causalgia is an extremely complicated, sympathetically maintained pain syndrome, which follows a small percentage of peripheral nerve injuries. While its exact pathophysiologic mechanism remains as yet unknown, a collusion of peripheral, central, and sympathetic nervous system activities initiate, maintain, and perpetuate the syndrome. Recent studies support the following hypothesis:

1. Initial peripheral nerve injury stimulates C-PMN fibers, which sensitize WDR spinal neurons.
2. Neurogenic inflammation triggers the firing of additional C-PMNs, which in turn sensitize WDR neurons.
3. Sensitized WDR neurons respond abnormally not only to C-PMN fiber input but also to A-beta fiber input. Thus, both noxious and nonnociceptive stimulation is perceived as hyperintense and painful.
4. Sympathetic adrenergic activity perpetuates WDR sensitization and abnormal firing by triggering the firing of A-beta fibers and also sensitizing and exciting C-PMN fibers.
5. Sympathetic sensitization, peripheral sensitization, and the escalation of nociceptor

involvement as soft tissue pathology develops, perpetuates the pain syndrome even after the initial injury has abated.

A solid understanding of all the potential elements involved may help guide a more rational approach to the management of this syndrome.

Diagnosis

While much of the literature alludes to the notion of CRPS as a unique, well-defined disorder, no studies to date have established firm criteria upon which a diagnosis can be based. Causalgia remains an elusive symptom complex, associated with laboratory findings that may help support but cannot confirm its presence. Most studies examining diagnostic approaches to CRPS do not differentiate Type I from Type II or SMP. Therefore, these terms may be used interchangeably in the next several sections.

Clinical Criteria

Descriptive definitions of CRPS-Type II and CRPS-Type I have been attempted. The IASP classifications for these symptom complexes have already been mentioned.[301] A panel of experts has attempted to develop a more concise definition. The group at Schloss Rettershoff[329] described CRPS-Type I as

> ...a syndrome of continuous diffuse limb pain, often burning in nature and usually consequent to injury or noxious stimulus and disuse, presenting with variable sensory, motor, autonomic, and trophic changes; causalgia represents a specific presentation of CRPS-Type I associated with peripheral nerve injury.[329]

While less vague than the 1986 IASP definition, detractors still lament that this definition of CRPS-Type I can apply to symptoms experienced in any number of posttraumatic situations.

The often-cited criteria established by Kozin et al[330] were developed empirically to assess the efficacy of certain treatment protocols and imaging studies. Kozin's proposed criteria divided patients into four potential groups:

1. Definite reflex sympathetic dystrophy syndrome (RSDS) included patients with pain, tenderness in the distal extremity, signs or symptoms of vasomotor instability, or swelling.
2. Probable RSDS included those with pain and tenderness and either vasomotor instability or swelling.
3. Possible RSDS included patients with vasomotor instability or swelling but without pain.
4. Lastly, the doubtful RSDS category contained those with unexplained pain in an extremity.

Ironically, while many clinicians base diagnoses on the responsiveness to sympathetic blockade, absolutely no patients in Kozin's study, even those deemed as suffering from definite RSDS, achieved a good response to sympathetic blockade. Kozin's scintigraphic results pose another problem when assessed in relationship to the duration of signs and symptoms. The expression of signs and symptoms by patients included in the study ranged from 2 weeks to nearly 6 years. Yet, from 40% to 83% had increased perfusion on the static and flow studies, varying merely by predetermined categorizations. These findings are at odds with the chronological evolution of bone scan findings established by Demangeat et al.[331] It should be understood, therefore, that the efficacy of Kozin's criterion as a diagnostic tool in establishing the diagnosis of CRPS-Type I is dubious at best.

An alternative approach to diagnosis was established by Roberts.[303] Of the signs and symptoms catalogued in other descriptions, only involvement of the sympathetic nervous system distinguishes the syndrome from other posttraumatic pain problems. A diagnosis of sympathetically maintained pain implies that pain relief is obtained during blockade of the sympathetic nervous system. Ganglionic blockade as well as postganglionic regional blockade have been considered. Arner[332] suggested that intravenous infusion of phentolamine, a short-acting alpha-adrenergic blocker (5 mg to 15 mg) over a 5-to-10-minute period could be used diagnostically to determine those patients likely to gain pain relief from more definitive sympathetic blockade. While narrow in focus, the establishment of responsiveness to sympathetic blockade may be exceedingly helpful in the orchestration of a multifaceted, interdisciplinary management approach.

Laboratory Findings

Roentgenography

Kozin et al[330] observed on fine detail roentgenography the nonspecific finding of patchy osteoporosis in 69% of patients meeting his criteria for definite, possible, and probable RSDS. Bone resorption was observed most commonly in the periarticular regions but also diffusely in a smaller number of cases. As a diagnostic tool, roentgenography has a sensitivity of 69% and a specificity of 79%.[330] Kozin and colleagues[333] cautioned that the development of patchy demineralization reflected the intensity of the resorptive process and not the actual mechanism. The value of roentgenography in their studies are overestimated in that associated problems, such as disuse and immobilization caused by nerve injury or pain, may be independent antecedents of periarticular osteoporosis.[334] The utility of roentgenography may be greatest in the very early stages when the resorption is less likely to be from disuse and more likely caused by accelerated blood flow in the affected region.

Triple-Phase Bone Scan

Several studies have addressed the utility of Technetium 99m (^{99m}Tc) bone scintigraphy as not only a diagnostic but a staging tool. Kozin et al noted the delayed phase (third phase) had a sensitivity of 60% and a specificity of 92%.[330,334] Holder and Mackinnon,[335] observing symptoms only in the hands, documented different levels of sensitivity during each of the three phases. Phase I had a sensitivity of 45%; phase II, 52%; and phase III, 96%. Specificity was virtually the same for all phases at greater than 95%. Davidoff et al[336] explored the predictive value of triple-phase bone scans. Overall sensitivity was rated at 44%, specificity was 92%, positive predictive value was 61%, and negative predictive value, 86%. Differences in outcomes likely reflect the lack of consensus on what constitutes a diagnosis of CRPS-Type I as well as the utilization of subtly different techniques. Other factors that have been noted to decrease the efficacy of triple-phase bone scanning as a diagnostic aid include the manifestation of symptoms for more than 6 months and patient age below 50 years.[337]

Demangeat et al[331] suggested that the variability of sensitivity might be explained if the period of illness during which the bone scan is obtained is considered. Following an initial intravenous bolus of ^{99m}Tc, the time span from the arrival of tracer in the camera field as it passes through the arterial system to the beginning of its disappearance through venous return marks the blood flow phase (phase I). The blood pool phase (phase II) follows quickly, revealing distribution of ^{99m}Tc between the intravascular and interstitial compartments. This phase reflects the vascularization of the soft tissues. By 3 hours after injection, the ^{99m}Tc has left the soft tissue. These delayed, phase III images reflect bone fixation (Figures 9-40 through 9-42).

Distinct patterns of ^{99m}Tc uptake during the three phases paralleled the elapsed time during which symptoms were evident.

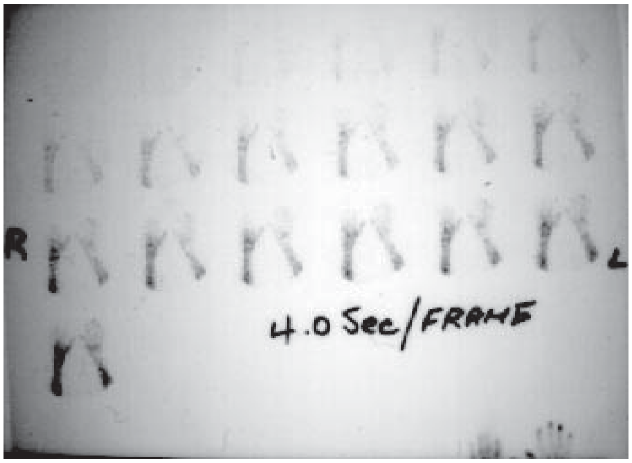


Fig. 9-40. Blood flow phase (phase I) of three phase bone scan. Asymmetrically increased uptake is observed in the left upper extremity of a patient with stage I CRPS-Type I. Scan courtesy of MAJ Antonio Balingit, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

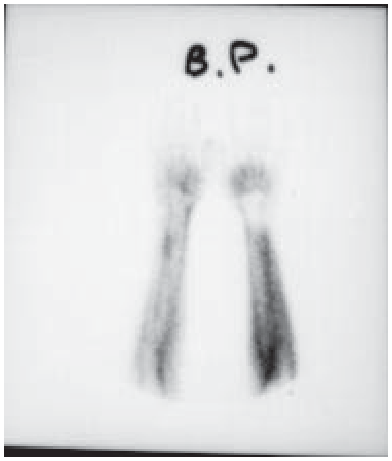


Fig. 9-41. Blood pool phase (phase II) of three phase bone scan. Asymmetrical increased uptake is observed in the right upper extremity of a different patient with stage I CRPS-Type I. Scan courtesy of MAJ Antonio Balingit, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

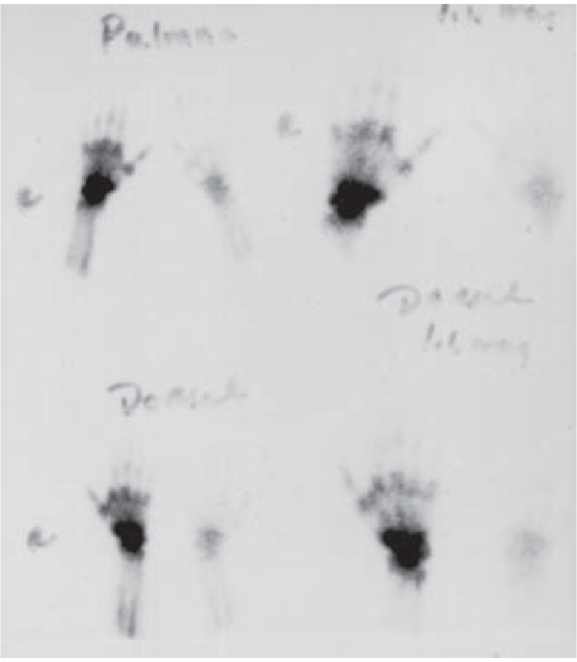


Fig. 9-42. Delayed hyperfixation phase (phase III) of three-phase bone scan. Asymmetrical periarticular increased uptake is observed in the right upper extremity of a patient with stage I CRPS-Type I. Tracing courtesy of MAJ Antonio Balingit, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

During the first 20 weeks from onset of symptoms, increased uptake was observed during all three phases. Normalization of the blood flow and pool phases were seen between 20 and 60 weeks with continued hyperfixation during phase III. After 60 weeks from onset, decreased uptake was observed in the first two phases with normalization in the last phase (Table 9-12).

Triple phase bone scan may be a helpful adjunct in the diagnosis of CRPS-Type I, especially in situations where symptoms, timeframe, and appropriate scintigraphic findings all coincide.

Thermography

Thermography is a noninvasive procedure that measures and displays heat emitted by the superficial tissues of the body. Normally, thermal distributions are symmetrical and follow a generally distinctive pattern. Changes in the digital color representations of temperature distributions or gradients may suggest an underlying pathology. According to Uricchio, thermographic findings in causalgia mirror clinical symptoms. Early in the course, an asymmetrical nonthermotomal temperature increase of several degrees is apparent by thermographic assessment. Later, as the limb becomes cooler, a regional drop in temperature may be seen in the affected limb.[338] While promising, the prohibitively high equipment costs and lack of general availability have kept thermography more in the realm of a research tool. There have been no studies comparing the benefits of thermography to the universally available and less costly scintigraphic studies.

Future Diagnostic Approach

Currently, there is no single test or group of findings that will absolutely confirm the diagnosis of CRPS. Further research is needed to establish the validity (ie, the test accurately measures what it intends to measure) of current diagnostic approaches. A paradigm shift needs to be made away from classification systems that require a person to meet all criteria in order to attain a diagnosis. The overall reliability (ie, capacity to give consistent results when used by different examiners at different times) of each diagnosis is limited by the least reliable criterion. The implementation of a model in which a

TABLE 9-12

TRIPLE PHASE BONE SCAN FINDINGS DURING THREE STAGES OF RSDS

	Bone Scan	
Stage	**Phase**	**Results**
Stage I (0–20 weeks)	Blood flow	Increased
	Blood pool	Increased
	Delayed	Increased
Stage II (20–60 weeks)	Blood flow	Normal
	Blood pool	Normal
	Delayed	Increased
Stage III (60–100 weeks)	Blood flow	Decreased
	Blood pool	Decreased
	Delayed	Normal

RSDS: reflex sympathetic dystrophy syndrome
Source: Davidoff G, Werner R, Cremer S, Jackson MD, Ventocilla C, Wolf L. Predictive value of the three-phase technetium bone scan in diagnosis of reflex sympathetic dystrophy syndrome. *Arch Phys Med Rehabil*. 1989;70:135–137.

person needs to manifest several, but not necessarily all, criteria will improve the overall reliability of each diagnosis and begin the process of honing a generally accepted approach to diagnosis. Such a model has been developed by Wilson[339] and may provide a basis for further diagnostic standardization. At least six criteria would be needed to confirm a diagnosis of CRPS-Type I. Three to five positive criteria may be categorized as possible CRPS-Type I until further evaluation of the criteria can be made. Less than three positive criteria would not constitute CRPS-Type I. Included as criteria would be both clinical and laboratory findings (Table 9-13).

Conclusions regarding the efficacy of specific treatment modalities are contingent on the development of valid diagnostic methods. Until criteria are accepted universally, it will be difficult to accurately assess the effectiveness of therapeutic interventions.

Treatment

No single treatment has been found to be effective in the treatment of CRPS-Type II. A well-coordinated, interdisciplinary approach with a focused goal of functional restoration is the key to successful management. The coupling of peripheral stimulation and central sensitization by sympathetic activity provides three areas that should be treated simultaneously to stymie the self-perpetuating cycle. Initial management should focus on the diagnosis and treatment of the inciting injury. Pain must be brought under control through peripheral, central, and sympathetic treatment approaches and, once decreased to tolerable levels, should be coordinated with aggressive physical therapy.

TABLE 9-13

PROPOSED DIAGNOSTIC CRITERIA FOR COMPLEX REGIONAL PAIN SYNDROME (CRPS)

Clinical Findings	Paraclinical Finding
Burning pain	Roentgenography
Allodynia	Bone scintigraphy
Temperature/color change	Thermography
Edema	Quantitative sweat test
Skin, hair, nail growth change	Quantitative sensory test
	Response to sympathetic blockade

Definite CRPS: More than 5 findings present
Possible CRPS: 3 to 5 findings present
Not CRPS: Less than 3 findings
Source: Wilson PR. Sympathetically maintained pain principles of diagnosis and therapy. In: Stanton-Hicks M, Janig W, Boas RA, eds. *Reflex Sympathetic Dystrophy*. Boston, Mass: Kluwer Academic Publishers; 1988:24–36.

Sympathetic Blockade

Extensive reports collected during World Wars I and II suggest that sympathetic blockade effectively abolishes the pain associated with CRPS-Type II.[316] As an isolated intervention, however, it rarely provides permanent relief.

Ganglionic blockade. Stellate ganglion blocks are used when the upper extremity is symptomatic. The needle is passed in a vertical-dorsal direction along the medial edge of the sternocleidomastoid muscle at the level of the cricoid cartilage. At a depth of 2 cm to 3.5 cm, contact should be made with the transverse process of the C-6 vertebra. The needle is then withdrawn 2 mm. A 5-mL to 10-mL injection of local anesthetic (eg, 1% mepivacaine or 0.5% bupivacaine) is administered following extensive aspiration, ensuring that no vessel has been entered inadvertently. Fluoroscopy may be exceedingly helpful to guide positioning.

Lumbar sympathetic blocks are used for lower extremity symptoms. The needle is passed in the medio-ventral direction, toward the L-2 vertebra. At 6-cm to 8-cm depth, the needle should come in contact with the body of the L-2 vertebra. The needle is withdrawn 1-cm to 2-cm and repositioned cephalo-laterally, placing it 1 cm lateral to the body of the L-1 vertebra. This approach reduces the risk of penetration into the kidney or psoas sheath. A 10-mL to 15-mL local anesthetic solution is injected. Blind technique carries a success rate of 80%. The use of contrast and fluoroscopic guidance improves the success rate to nearly 100%.[340]

An adequate sympathetic blockade should cause a temperature rise in the affected limb of 5ºC to 10ºC. A Horner syndrome is a common side effect of a successful stellate ganglion block.[341]

Regional blockade. Postganglionic sympathetic blockade can also be achieved through regional intravenous administration of sympatholytic agents.

Guanethidine infusion was introduced in 1974 by Hannington-Kiff[342] and remains the gold standard. Intravenous administration has not been approved in the United States. Alternatives including reserpine and bretylium have also been employed with similar results.[343,344]

Similar results have been achieved with both ganglionic and regional blockade. Bonelli et al[345] observed that symptomatic pain relief and evidence of sympathetic blockade following intravenous regional guanethidine administration every 4 days for a total of 4 blocks was comparable to stellate ganglion blocks performed daily for 8 days. The major advantage of intravenous regional blockade can be seen during treatment of CRPS-Type II in the upper extremity. The ease of intravenous administration easily outweighs the potential hazards of stellate ganglion blockade, which include pneumothorax, intravascular injection, and puncture of the esophagus. The opposite techniques are more easily used in the lower extremity. Lumbar sympathetic blockade is a relatively easy procedure, while tourniquet placement and venous cannulation during regional blockade pose problems in the lower extremity.[346]

Oral Administration

A single uncontrolled study and an anecdotal report[347] discuss the use of oral sympatholytic agents in the treatment of causalgia. Ghostine administered phenoxybenzamine, a long-acting peripheral alpha$_1$-adrenergic blocker, to nerve-injured soldiers with CRPS-Type II during the Lebanese civil war. Initial dosing started at 30 mg per day in three divided doses (ie, 10 mg tid). The dose was increased by 10 mg every 2 days until symptomatic relief was achieved or side effects became unbearable. The average dose was 80 mg daily. The treatment was continued for 6 weeks in the majority of patients with excellent, long-lasting results.[347]

Sympathectomy

In patients who respond to sympathetic blockade, but only transiently, and do not tolerate oral sympatholytic agents, permanent sympathectomy may be considered. Bonica[316] suggests that chemical sympathectomy with 6% aqueous phenol or 50% alcohol can ablate sympathetic activity for several months. It was suggested that this procedure might be better tolerated in older patients than the surgical procedure. Surgical sympathectomy might be tried in younger, healthier patients. Both Bonica[316] and Mayfield[304] warn, however, that sympathectomies rarely provide permanent, long-lasting pain relief. If undertaken, they still must be coordinated with other methods of pain modulation.

Pain relief derived from sympathetic blockade provides three major benefits in the treatment of CRPS-Type II. Aside from direct enhancement of patient comfort, it allows the patient to use the affected limb. It also establishes the diagnosis of sympathetically maintained pain, differentiating the syndrome from other forms of neuropathic pain.

Peripheral Treatment

Injury Management

Injury to peripheral nociceptors and persistent stimulation of nociceptors and mechanoreceptors perpetuate the symptom complex. Diagnosis and rapid treatment of the inciting injury are paramount in the management of CRPS-Type II.

Pain Control

The use of pain-modulating agents that act peripherally has been advocated. While few controlled trials have established their effectiveness, empiric usage stems from results achieved in the treatment of other types of nerve injury.

Corticosteroid administration was found to be highly effective in the Kozin et al study[348] for patients meeting his criteria of definite RSDS. A randomized, placebo-controlled study by Christensen et al[349] also showed improvement beyond that attained with placebo. Thirty milligrams of prednisone was administered daily in divided doses for upwards of 12 weeks. Improvement was noted in all patients.

Nonsteroidal antiinflammatory agents have been included in review articles as potentially effective agents during the acute stage.[350] No controlled studies were noted to have been attempted to establish efficacy.

Case reports attesting to pain relief provided by topical application of clonidine[351] and capsaicin[352] have recently been cited, but lack adequate placebo-controlled trials. Oral administration of membrane stabilizers such as phenytoin,[353] beta-adrenergic blockers including propranolol,[354] gabapentin,[355] and calcium channel blockers have also been reported.[356]

Transcutaneous electrical nerve stimulation and electroacupuncture application have been shown in several small uncontrolled trials to be effective in the diminution of CRPS-Type II-related pain.[357,358]

Physical Modalities

It has been suggested that, ultimately, the greatest benefit of pain management is that it allows the patient to participate in therapies that will restore normal function. Edema control and the resolution of contractures decrease stimulation of peripheral mechanoreceptors and nociceptors. Repetitive contraction of muscles during progressive resistance training helps to decrease edema accumulation and reverses osteoporotic bony changes. Hypesthesia, a common manifestation of CRPS-Type II, can be effectively dampened through desensitization techniques. While research examining the exact benefits of physical modalities has been severely limited, there is widespread agreement that they are an integral part of the treatment program.[356] All efforts need to be made to encourage use of the affected limb as much as possible.

Central Treatment

Descending central-pain-modulating pathways provide inhibitory impulses at the level of the dorsal horn and may provide an element of pain relief. The use of drugs including tricyclic antidepressants have been recommended despite the lack of clinical trials.[341,356]

As with other chronic pain syndromes, the distress and anxiety caused by the experience of persistent suffering impairs the ability to cope not only with the pain syndrome but also the stressors of day-to-day life. Intense psychologic support is essential and all efforts should be made to allay fears and anxieties about using the affected limb. Affective disorders, which may arise secondarily, should be monitored closely for detection and treated appropriately.[316]

Case Study 1: Penetrating Injury to the Upper Extremity

The patient is a 46-year-old army sergeant. In February 1991, he was struck by an artillery shell fragment which entered his left upper arm, fracturing the humerus and severing the brachial artery. He underwent emergent vascular reconstructive surgery at the evacuation hospital in Saudi Arabia. At the time he was observed to have complete median and radial nerve injuries of the left arm. Surgical clips were attached to the ends of these nerves, marking them for potential nerve grafting in the future. Open reduction and internal fixation was performed to stabilize the humeral fracture.

The Physical Medicine and Rehabilitation Service was consulted on his transfer to Walter Reed Army Medical Center. Initial physical examination of his left upper extremity revealed marked edema, healing surgical wounds, no evidence of infection, and tenderness to palpation. Radial pulse was intact.

His passive shoulder range-of-motion was limited in abduction, internal rotation, and external rotation. Elbow flexion passively was limited to 70° with a firm endpoint. Passive flexion and extension at the wrist, MCP flexion and interphalangeal (IP) joint flexion were limited in all fingers in the left hand.

Strength was good in the proximal shoulder girdle muscles, poor in elbow flexion, and trace in elbow extension. Wrist flexion strength was fair with ulnar deviation observed at the wrist. Wrist extension and MCP joint extension in all fingers was zero as was thumb abduction, extension, opposition, and IP joint flexion. First dorsal interosseus muscle strength was graded as poor. Thumb abduction was good. Distal IP joint flexion was zero in the second digit, trace in the third digit, and fair in the fourth and fifth digits.

Sensation testing to light touch and pinprick revealed absent sensation over the palmar and dorsal aspect of the thumb and second and third digits. Mild sensory loss was also noted in the medial antebrachium. Deep tendon reflexes in the left upper extremity were absent. The patient also complained of severe pain, allodynia, and hypesthesia in the forearm and hand, exacerbated by passive motion and light stroking of the hand and forearm. The distal extremity was cool and cyanotic when compared to the opposite limb.

Plain radiographs of the left elbow and humerus revealed exuberant bony callous formation with heterotopic ossification and a stable, healing fracture. Electrodiagnostic testing 1 month postinjury revealed findings consistent with a severe median nerve injury at the elbow with no evoked potentials obtainable. Electrodiagnostic testing confirmed that the lesion was at a point proximal to the point where the nerve to the flexor carpi radialis arises. Radial motor nerve conductions were not obtainable, suggesting severe axonal injury. Radial SNAPs were obtained, revealing a decreased amplitude but normal latency. Coupled with the clinical finding of absent sensation in the radial distribution, the findings suggested mild to moderate axonal loss with severe conduction block. It was suspected that the focus of injury was likely just distal to the branching point of the superficial radial nerve. A partial, moderately severe left ulnar nerve injury with evidence of axonal loss was also observed. There was also evidence of a mild brachial plexopathy with spontaneous activity in the deltoid and teres minor muscles but with full recruitment. In addition, there was marked soft tissue injury in the brachial region involving the biceps and triceps which complicated electrodiagnostic interpretation.

Triple phase bone scan revealed diffuse increased uptake in the periarticular regions of the left hand and wrist suggestive of CRPS-Type I. In light of his symptoms and associated nerve injuries, a diagnosis of CRPS-Type II was made.

Improving and maintaining adequate functional range-of-motion in his left hand and arm required extensive therapy, including prolonged stretching by occupational therapists and a self-program that the patient followed diligently. The humerus fracture stabilization was strong enough to allow careful, active, assistive range-of-motion at the elbow. Initially, a static dorsal orthosis was fabricated and designed to provide an alternating MCP extension force and finger flexion force. The patient alternated the flexion and extension every few hours to provide prolonged stretch to the MCP and IP finger joints. His range-of-motion in the wrist and hand improved enough to allow fabrication of a functional dynamic orthosis.

Edema of the hand and forearm was treated initially with pneumatic compression followed by the application of a pressure garment sleeve and glove. Centripetal massage and wrapping were also employed. Significant improvement occurred over the course of several weeks.

Muscles not completely denervated—the ulnar innervated muscles, the triceps, the biceps, and the proximal arm and shoulder muscles—were exercised with active assistive, then active resistive exercises. The ulnar innervated muscles (interossei) improved markedly to a level of good (4/5). Good strength in wrist flexion, and good distal IP joint flexion of the fourth and fifth fingers was obtained. Elbow flexion and extension strength had improved to a level of good. Active pronation and supination remained zero. Metacarpophalangeal joint flexion strength in the second through fifth fingers improved to a good level. The proximal shoulder muscle strength improved to normal.

Pain management included administration of the NSAID Naprosyn, as well as the tricyclic antidepressant amitriptyline. The latter was stopped because of anticholinergic side effects. Doxepin was administered as an alternative. Conventional TENS was applied to the proximal aspect of the forearm, providing moderate pain relief. Desensitization, employing progressively harsh textures and vibration, helped to decrease the allodynic and hypesthetic sensations. These treatments in conjunction with reduction in edema and soft tissue contractures decreased the patient's pain to a tolerable level, allowing him to sleep soundly and perform daily tasks.

Sensory reeducation techniques were instituted to enhance the patient's dexterity and sensory discrimination in the ulnar distribution.

A dynamic wrist-hand orthosis was fabricated to provide stabilization and dynamic substitution for movements lost secondary to the multiple nerve injuries. The base orthosis consisted of a dorsal orthoplast plate covering two thirds of the dorsal aspect of the forearm. Wrist extension was achieved through the placement of a hinge at the wrist. Elastic bands provided dynamic extension assistance while allowing for active flexion, driven by the flexor carpi ulnaris. Extension assists were also incorporated, providing dynamic MCP extension for the second through fifth fingers. The extension assists consisted of finger loops attached by nylon cords which ran through eyelets at the distal end of the orthosis. The cords were attached to elastic bands, which were attached to the orthosis with Velcro, allowing for changes in desired tension. A coupling device yoked the second and third fingers, which allowed the third finger to power a three-chuck-jaw grasp. A small hook was attached to the coupler, which was connected to the main dorsal orthosis to provide dynamic extension assistance. A removable sleeve was fabricated to stabilize the thumb. It also was attached to the base orthosis by elastic bands and provided dynamic abduction and opposition. A volar plate provided the base to which the elastic thumb abduction assist was attached (Figures 9-43 and 9-44).

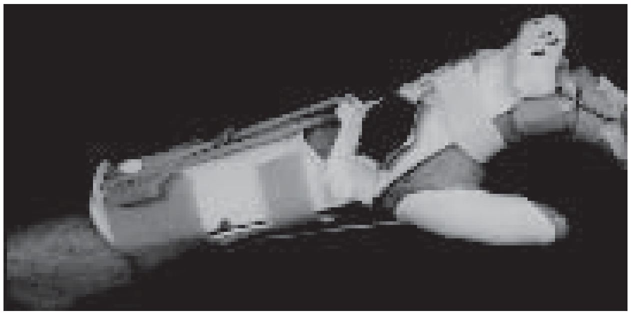


Fig 9-43. Profile view of dynamic wrist-hand orthosis that provides stability and dynamic assistance for the upper extremity of a patient who sustained complete radial and median nerve injuries and a partial ulnar nerve injury.

Extensive patient education and training were required to optimize the patient's hand function with the orthosis.

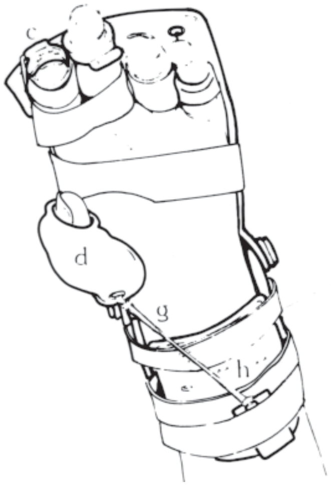


Fig 9-44. Palmar view of the orthosis. **c**: Coupling device yoking the second and third fingers. **d**: Thumb stabilizing sleeve. **g**: Elastic band providing dynamic thumb abduction and opposition. **h**: Volar plate providing base for dynamic thumb assist.

Routine skin inspection was essential to evaluate insensate skin for breakdown. The patient was able to grasp objects and use the orthosis to complete tasks requiring two hands, which he was unable to do without the orthosis.

Six months following his injury, the patient underwent interpositional sural nerve grafting of both the left median and radial nerves. At 2 years following the injury, there was no evidence of return of function or sensation in the distributions of the grafted nerves. During this period, however, the patient continued to use the orthosis while at work and during recreational activities. His pain had subsided and strength in the ulnar distribution had improved to normal levels. With this improvement in strength it was felt that tendon transfers could now be undertaken to improve the stability of the wrist and improve his function.

Transfer of the flexor carpi ulnaris tendon to the extensor digitorum communis and the extensor pollicis longus tendons provided finger and thumb extension. Next, the flexor digitorum profundus III tendon was transferred to the flexor digitorum profundus II tendon to improve index finger flexion. The thumb was stabilized by fusion of the IP joint. Wrist stabilization was achieved by performing an extensor carpi radialis brevis tenodesis.

Further therapy, including maintenance of range-of-motion, strengthening, and muscle reeducation, was necessary following surgery. The patient is currently functional using the left upper extremity.

Case Study 2: Penetrating Injury to the Lower Extremity

The patient is a 20-year-old army specialist. In October 1993, he was struck in the left popliteal region by fragments after his vehicle was hit by a rocket-propelled grenade. The patient was hemodynamically stabilized in Somalia and transferred to Landstuhl Army Medical Center in Germany. Surgical debridement of burned and necrotic tissue was undertaken and a split-thickness skin graft was placed to cover the popliteal region. The patient was then transferred to Walter Reed Army Medical Center for further evaluation and treatment. Upon arrival he was observed to have an ongoing soft-tissue infection and necrosis of the skin graft. The area was sharply debrided. The posterior tibial nerve was burned and contused (Figure 9-45). The common peroneal nerve was avulsed from the sciatic nerve at the bifurcation. The anterior tibial artery was ligated and the popliteal artery was observed to be in continuity but greatly inflamed. Five days later, the patient underwent a left latissimus dorsi free muscle flap to the left popliteal fossa with split-thickness skin grafting.

The Physical Medicine and Rehabilitation Service was consulted upon the patient's transfer to Walter Reed Army Medical Center. Initial physical examination of his left lower extremity, 10 days following surgery, revealed partial thickness burns to the distal medial aspect of the thigh. A 14 x 6 x 16-cm latissimus dorsi musculocutaneous flap covering the popliteal region was intact, edematous but healing well. Capillary refill at the toes was brisk.

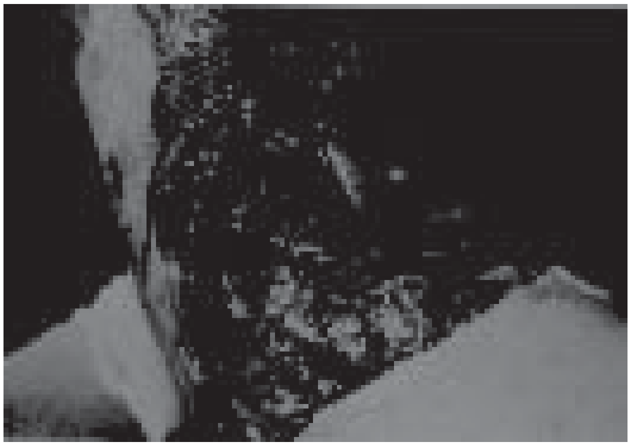


Fig 9-45. Surgical debridement of popliteal region following penetrating and burn injury. The tibial nerve is exposed and observed to be badly burned and contused. Photograph courtesy of LTC Gregory A. Antoine, M.D., Plastic and Reconstructive Surgery Service, Walter Reed Army Medical Center, Washington, DC.

Passive range-of-motion at the hip was limited to 90°. Knee flexion was limited to 30°. Knee extension was limited actively by 15° and could not be tested passively because of the recent graft placement. Passive ankle dorsiflexion was limited to neutral.

Strength was good in the proximal hip girdle with the exception of hip extension, which was fair. Knee flexion and extension were fair and significantly limited by pain. No active movement could be observed in ankle dorsiflexion, plantarflexion, inversion, eversion, or toe movement in any direction.

Sensation testing to light touch and pinprick revealed absent sensation along the lateral aspect of the leg, extending from just lateral to the crest of the tibia, posteriorly to the medial third of the posterior aspect of the calf. Sensation was also absent over the entire foot. The findings suggested loss of sensation in the sural, superficial peroneal, deep peroneal, and plantar nerves with sensory sparing in the saphenous nerve distribution (Figure 9-46). Deep tendon reflexes could not be tested at the left patellar tendon and were absent at the left Achilles tendon. The patient also complained of pain not only in the operative site but described a lancinating pain shooting down the back of the thigh and into the toes. He also described a burning, aching sensation in the anesthetic region of the left foot, which felt as though a vise was around his ankle, and his toes were in a contorted posture.

Plane radiographs of the entire left lower extremity revealed no evidence of fractures or joint dislocations. Arteriogram of the left lower extremity revealed no evidence of vascular injury.

Electrodiagnostic testing performed 6 weeks after the injury revealed findings consistent with severe common peroneal and tibial nerve injuries. Evoked potentials could

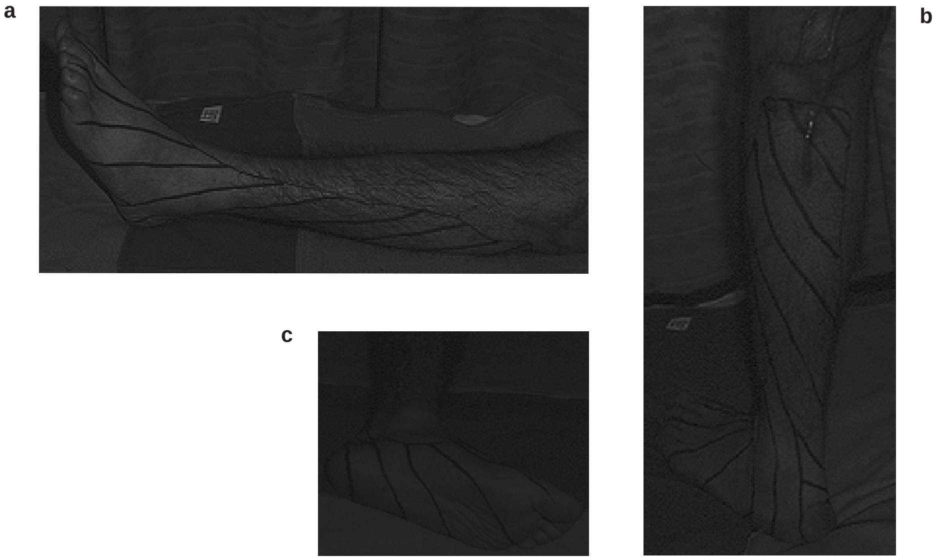


Fig 9-46. a: Anterolateral view. **b:** Sole of the foot. **c:** Posterolateral view. Demarcation of sensory loss following injury to the left common peroneal and tibial nerves. Absence of sensation was observed along the sural, superficial peroneal, deep peroneal, and tibial plantar nerves. Sensation in the saphenous nerve distribution was intact.

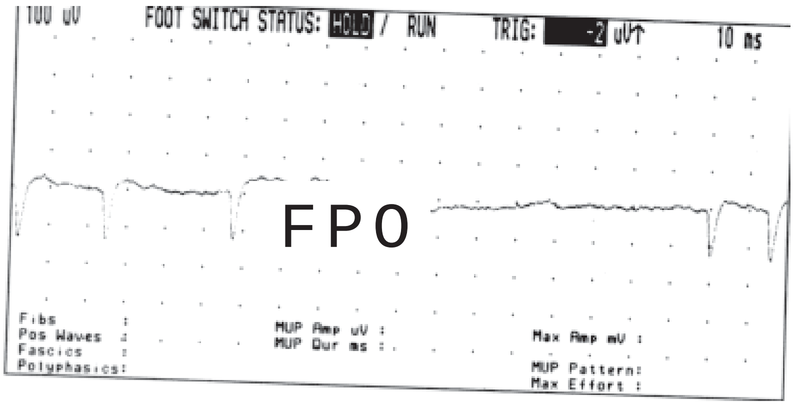


Fig 9-47. Electromyographic display of positive wave potentials observed during assessment for spontaneous activity. Examination was of the left medial gastrocnemius muscle.

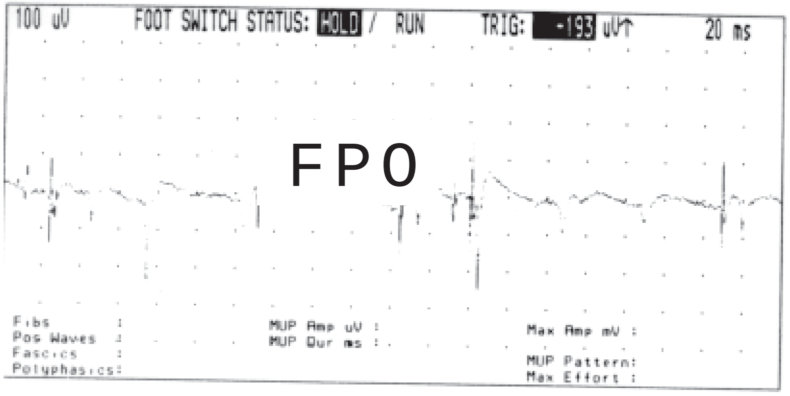


Fig 9-48. Electromyographic display of both positive sharp wave and fibrillation potentials observed during assessment for spontaneous activity. Examination was of the left tibialis anterior muscle.

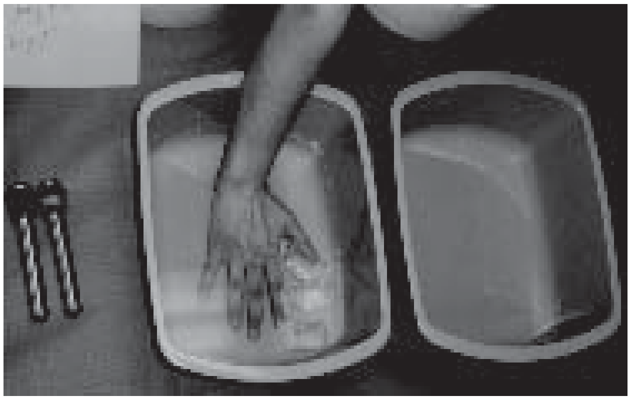


Fig 9-49. An example of contrast baths that are used to produce hyperemia and to decrease hypersensitivity to hot or cold temperatures. The hot bath is maintained between 40ºC and 43ºC. The cold bath temperature ranges between 15ºC and 20ºC. Immersion protocols include hot bath for 10 minutes, cold bath for 1 minute followed by cycles of 4-minute hot baths alternating with 1-minute cold baths. This is continued for a full 30-minute session. Adapted with permission from Lehmann JF, deLateur BJ. Diathermy and superficial heat, laser and cold therapy. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* 4th ed. Philadelphia, Pa: WB Saunders; 1990: 283–367.

not be obtained during near nerve stimulation proximal and distal to the site of injury. Saphenous sensory conduction was normal. Electromyography revealed evidence of severe, ongoing denervation in muscles of the calf and foot innervated by the superficial peroneal, deep peroneal, and tibial nerves (Figure 9-47 and 9-48). More proximal evaluation suggested the focus of injury to be distal to the branch to the short head of the biceps femoris muscle. There was no evidence to suggest a concomitant plexus or root injury.

Pain management became the major issue following surgery. Trials of ketorolac and meperidine postoperatively did not adequately relieve the patient's pain. The pain precluded his ability to transfer out of the bed, to ambulate or to attempt ADLs. It was recommended that amitriptyline be started, titrating from 25 to 150 mg every night. This was later changed to trazadone 100 mg nightly because of anticholinergic side effects. It was also suggested that Demerol be stopped because of its potential for provoking seizures, and a long-acting morphine sulfate agent to be started at 30 mg twice daily (bid). The patient continued to complain of lancinating pain. The long acting morphine sulfate was increased to 30 mg tid. The NSAID Naprosyn at 500 mg bid was added, as was carbamazepine at a dose titrated up to 200 mg tid, which significantly improved the patient's lancinating pain. The patient was switched to Methadone and gradually weaned off all narcotics over a several-week period without a decrease in pain control. Conventional TENS was applied to the proximal aspect of the thigh, which provided only minimal relief and was subsequently stopped. Desensitization techniques including progressive textures and vibration, and contrast baths helped to decrease the abnormal perception of his toes and ankle (Figure 9-49). Better control of neuropathic and deafferentation pain allowed the patient to participate more aggressively in his therapies.

A plastic molded ankle-foot orthosis was fabricated to provide ankle stabilization and to simulate dorsiflexion and plantar flexion. Trim lines were brought anteriorly to provide mediolateral stability. Medial arch support and lateral flare were built into the foot plate to help stabilize and support the foot. The foot plate was extended to just distal to the metatarsal heads to prevent collapse of the mid-foot and simulate pushoff at the end of the stance phase. The ankle was set in 5° of plantarflexion, which allowed for adequate toe clearance during the swing phase. It also allowed for the most normal forces to be generated at the knee during the stance phase, averting hyperextension or a destabilizing knee flexion moment (Figure 9-50). Extensive gait training was required to optimize the patient's ability to ambulate with the orthosis. Scrupulous skin monitoring was necessary and taught to the patient, as his protective sensation was limited to the saphenous distribution.

After the patient was cleared from the plastic surgery service, active assistive and passive range-of-motion ex-

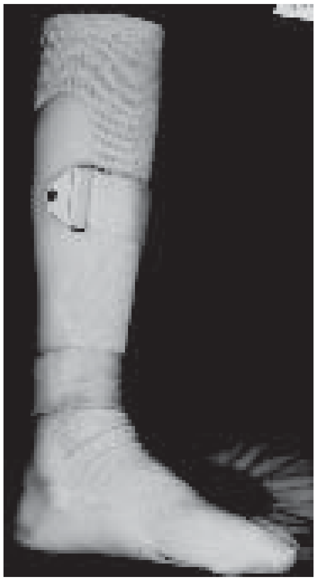


Fig 9-50. Left copolymer, custom molded ankle foot orthosis. The trim lines at the ankle are reinforced, providing mediolateral stability. The ankle is set in 5° platarflexion, allowing toe clearance and knee stability. The foot plate is extended just distal to the metatarsal heads to provide simulated pushoff at the end of the stance phase in the gait cycle.

ercises were instituted. Moist heat application around the left knee was used in conjunction with slow, prolonged stretching. By 10 weeks following his injury, the patient had regained full passive range-of-motion at the knee, hip, and ankle. Active assistive stretching of the shoulder flexors and external rotators was also done to maintain full mobility following harvesting of the latissimus dorsi musculocutaneous flap.

Edema of the lower extremity was treated with centripetal massage and wrapping. Resolution of swelling occurred over the course of several weeks. Compression garments were fabricated not only to maintain edema control but also to decrease hypertrophic scarring which developed in burned regions of the leg.

Progressive resistive exercises of all muscles in the lower extremity with strength of at least fair (3/5) led to marked improvement in strength. By 3 months postinjury the patient had regained normal strength about the hip girdle and knee.

General aerobic training, including cycle ergometry and swimming, improved the patient's endurance to a level at which he could maintain activity at 70% of his calculated maximum heart rate for 45 to 60 minutes.

The patient was reevaluated by the plastic surgery and orthopedic surgery services 4 months following injury. It was suggested that attempts at nerve grafting would not likely be beneficial and were not attempted. The possibility of triple arthrodesis to stabilize the ankle was entertained. The procedure was deferred by the patient who expressed that he was not limited functionally at the time, was experiencing only minimal discomfort in anesthetic regions, and was fully functional using the ankle foot orthosis.

Five months following the injury, the patient was fully independent and had been taken off all medication with the exception of trazadone, which helped relieve his deafferentation pain experienced in the evenings, allowing him to sleep soundly.

UPPER EXTREMITY NERVE INJURIES AND ENTRAPMENT SYNDROMES

Brachial Plexopathies

Anatomic Considerations

Brachial plexopathies are defined as nerve lesions involving any part of the peripheral nervous system distal to the nerve root and proximal to the main nerve branches. Thus, brachial plexopathy refers to postganglionic nerve pathology. The nerve lesion in brachial plexopathies occurs distal to the cell bodies of both the motor and sensory nerve fibers.

The fifth and sixth cervical nerve roots unite between the scalenus medius and scalenus anterior to form the upper trunk of the brachial plexus. The seventh cervical nerve root courses behind the lateral margin of the scalenus anterior to form the middle trunk. The eighth cervical and first thoracic nerve roots unite behind Sibson's fascia with the neck of the first rib between the two spinal nerves. Together they form the lower trunk, which emerges lateral to the fascia (Figure 9-51).

The upper, middle, and lower trunks divide into anterior and posterior divisions lateral to the first rib. The anterior divisions of the upper and middle trunk form the lateral cord and the posterior divisions form the posterior cord. The anterior division of the lower trunk forms the medial cord.

The cords then divide into their respective terminal branches. The lateral cord divides into the musculocutaneous nerve and the lateral head of the median nerve. The medial cord divides into the ulnar nerve and medial head of the median nerve. The posterior cord separates into the radial and axillary nerves. The long thoracic, subclavius, and dorsal scapular nerves branch off directly from the cervical nerve roots.

The long thoracic nerve is derived from nerve fiber contributions from the fifth, sixth, and seventh cervical nerve roots and innervates the serratus anterior muscle. The subclavius nerve arises from the fifth and sixth cervical nerve roots to serve the subclavius muscle. The dorsal scapular nerve is a branch of the fifth cervical nerve root and innervates both the rhomboids and the levator scapulae muscles.

The suprascapular nerve is the sole motor nerve branch emerging from the upper trunk shortly after its formation. It innervates the supraspinatus and infraspinatus muscles.

The subscapular, thoracodorsal, lateral pectoral, medial pectoral, and medial cutaneous nerves of the arm and forearm emerge from the cords of the brachial plexus. The subscapular nerve branches off the posterior cord to innervate the subscapularis and teres major. The thoracodorsal nerve also divides from the posterior cord to serve the latissimus dorsi muscle. The lateral pectoral nerve, a branch of the lateral cord, innervates portions of the pectoralis major and minor muscles. The medial pectoral nerve is a branch off the medial cord. It also contributes nerve fibers to the pectorals major and minor muscles. The medial cutaneous nerves of the arm and forearm are derived from the medial cord and innervate the medial arm and forearm, respectively.

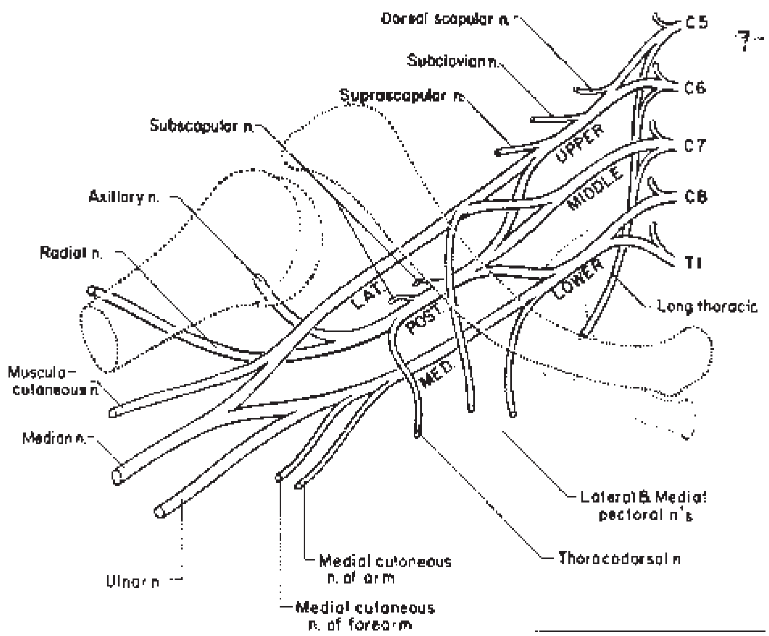


Fig. 9-51. The brachial plexus. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 40.

Etiology

Numerous clinical conditions are associated with plexopathies with an equally wide range of clinical presentations. These clinical conditions include arteriovenous fistula,[359] postradiation therapy,[360,361] postmedian sternotomy,[362–364] Parsonage-Turner syndrome,[365,366] polyarteritis nodosa,[367] viral infections (mononucleosis[368] and parvovirus[369]), herpes zoster,[370] trauma,[371,372] superior sulcus tumors,[373] and coagulopathies.[374]

Brachial plexus injuries may occur at any level and involve a variable degree of the plexus. The majority of lesions, in fact, are not uniform and various degrees of nerve damage typically coexist as a result of an injury.[9]

Brachial plexus injuries may be the result of wounds from knives and other penetrating objects, lacerations, bullet wounds, and shell fragments. The supraclavicular aspect of the brachial plexus is more prone to the latter injuries resulting in primary insult to the upper and middle aspect of the plexus. Lower plexus lesions may concomitantly involve insult to the lungs or great vessels. Thus, individuals with injury to this portion of the plexus are at significantly greater risk of sustaining a fatal wound.

Interestingly, the brachial plexus remains in continuity in a large percentage of open brachial plexus missile and shell fragment wounds.[9] Nelson et al[375] reported on nine cases during the Vietnam War in which patients sustained brachial plexus injury secondary to severe missile wounds of the chest. They found that all of these patients subsequently had rapid, spontaneous recovery of their plexus injury. It should be noted, however, that more severe injuries to the plexus may likely have been associated with significant vascular injuries. Disruption of the subclavian or axillary arteries in a combat setting likely would end in exsanguination. Thus, the authors may have observed only less severe injuries.

Brachial plexus injury can occur as a result of operative intervention due to improper retraction or inadvertent transection, clamping, or ligation of nerves. For example, median sternotomy performed as part of intracardiac surgery may damage the brachial plexus.[376] It may be iatrogenically induced by axillary puncture performed for arteriography as well.[377,378] Closed brachial plexus lesions may result from direct blows or prolonged, excessive pressure applied to the supraclavicular region. It most commonly occurs, however, as a secondary complication. Thus, the plexus can be lacerated by the edge of a fractured bone; by a sharp bone fragment; or from compression by dislocated or fractured bone, aneurysm, or hematoma.[9]

Brachial plexus injury has been documented in a variety of sports activities. Upper trunk damage, for example, has been recorded in hockey and lacrosse due to severe blows directed at the angle between the neck and shoulder.[379] A rifle's recoil has been reported to cause plexus injury by pressing the clavicle against the upper trunk.[380] Rucksack palsy occurs as a result of upper trunk injury from direct downward pressure on the plexus or by trapping the plexus between the clavicle and the deeper structures, or both.[381] Traction injuries most commonly involve the upper plexus. Wynn Parry[382] reviewed multiple brachial plexopathies attributable to traction injury. He discovered that the upper plexus was 10 times more frequently injured vs the lower plexus. He also found that the great majority of injuries in his series were due to motorcycle accidents, followed by car accidents.

Clinical Presentation

Upper trunk injuries generally result in characteristic deficits. In complete lesions, the arm hangs at the side in an adducted and internally rotated posture. Atrophy is evident in the shoulder abductors, external rotators, and extensors. It is also found in the forearm flexors and supinators. Elbow extension and forearm pronation, as well as wrist and finger flexion strength are essentially preserved. Sensation to light touch and pinprick is generally diminished on the outer aspect of the arm and forearm, extending to the radial side of the hand.

Middle trunk damage occurs only infrequently as an isolated injury. Insult to the middle trunk will result in decreased elbow extension, as well as wrist and long-finger extensor weakness. Sensory deficits occur in the distribution of the C-7 nerve root.

Lower trunk injuries result in atrophy and weakness of the intrinsic muscles of the hand, and less prominent atrophy of the forearm flexor region as well. Sensory impairment is manifested by diminished cutaneous sensation from the medial aspect of the arm at a point just about the elbow extending to the ulnar aspect of the hand and little and ring fingers. In severe injuries, a complete plexus injury or panbrachial plexopathy may occur. In this case the patient presents with complete flaccid paralysis and diffuse loss or impaired sensation of the involved extremity. The patient may also have an ipsilateral Horner's sign consistent with compromise of the sympathetic ganglion at the C-8, T-1 level, as well as vasotrophic changes, particularly involving the distal aspect of the limb.

Injury to Nerve Branches of the Brachial Plexus

Long Thoracic Nerve

Nerve branches of the brachial plexus may be selectively injured. The long thoracic nerve may be damaged by electric shock and trauma, or it may occur as an idiopathic phenomenon (Figure 9-52).[383,384] Long thoracic nerve palsy has been documented to occur in temporal relationship to serum and vaccine injections.[385,386]

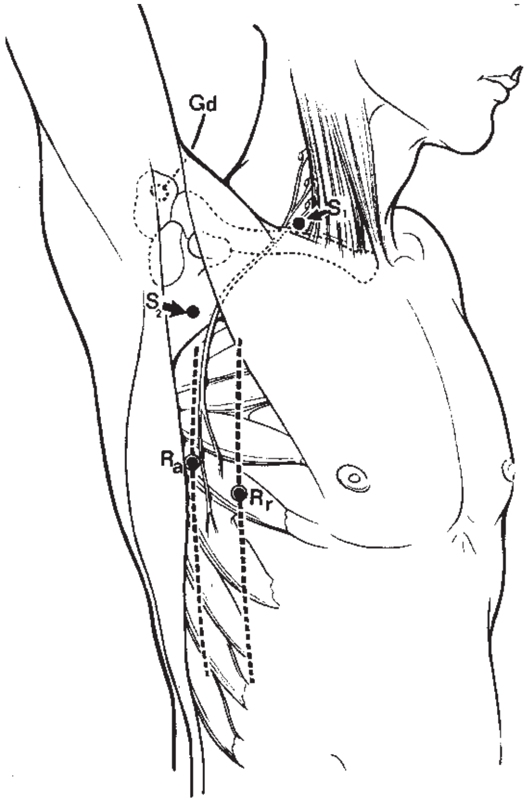


Fig. 9-52. The long thoracic nerve with motor nerve conduction electrode placement. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 48.

Suprascapular Nerve

Rarely, the suprascapular nerve is entrapped in the suprascapular notch(Figure 9-53).[387–390] The suprascapular nerve may be entrapped at the level of the spinoglenoid notch, resulting in isolated weakness of the infraspinatus muscle.[391,392] It may be specifically injured secondary to compression by ganglia at the spinoglenoid notch.[393]

Medial Antebrachial Cutaneous Nerve

The medial antebrachial nerve injury has occurred as a complication of surgical repair of a cubital tunnel syndrome.[394] It also may be damaged as a consequence of a stretch injury (Figure 9-54).[395]

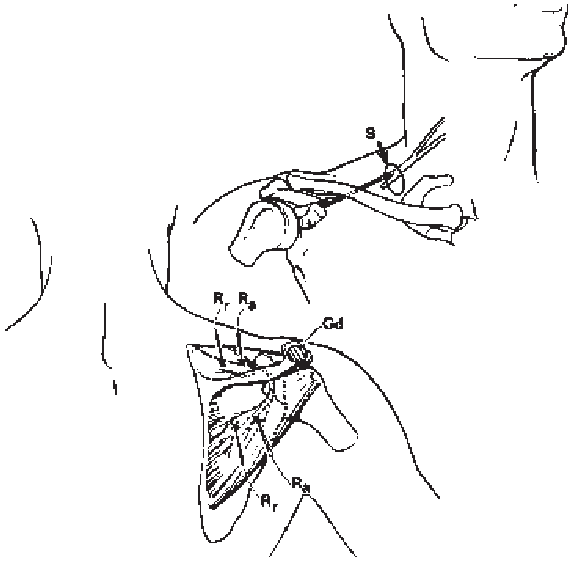


Fig. 9-53. The suprascapular nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 55.

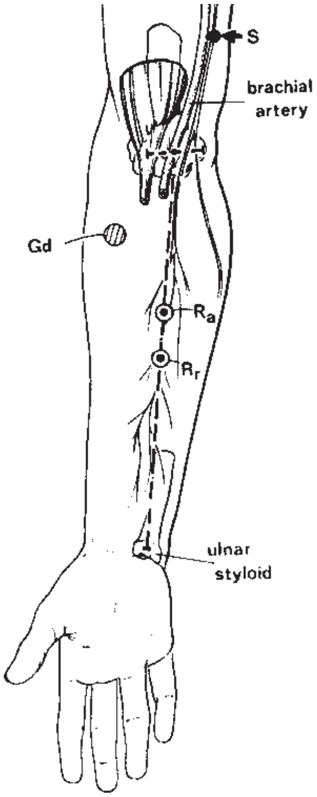


Fig. 9-54. The medial antebrachial cutaneous nerve with electrode placement for sensory nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985:147.

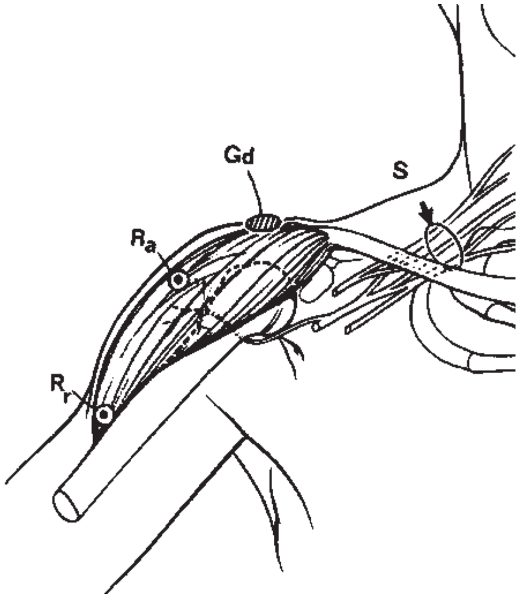


Fig. 9-55. The axillary nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 68.

Axillary Nerve

Isolated axillary nerve injury may occur following humeral head fractures, posterior dislocations or manipulations (Figure 9-55).[396,397] It may also occur after intramuscular injection into the posterior shoulder area.[398] Shoulder dislocation may also cause injury to the posterior cord, resulting in weakness in muscles innervated by the axillary and radial nerves (Figure 9-56).[399]

The axillary,[400] long thoracic,[401] and musculocutaneous nerves[402] may be injured secondary to surgical positioning or as a complication of obstetrical deliveries.

Musculocutaneous Nerve

The musculocutaneous nerve may be lacerated as a complication of a midshaft humeral fracture[403] or entrapped by the biceps aponeurosis and tendon at the level of the elbow during exercise activities.[404]

Electrodiagnosis of Brachial Plexopathy

One of the most important challenges an electromyographer encounters in evaluation of a brachial plexopathy is to distinguish between pre- and postganglionic lesions. In isolated preganglionic lesions,

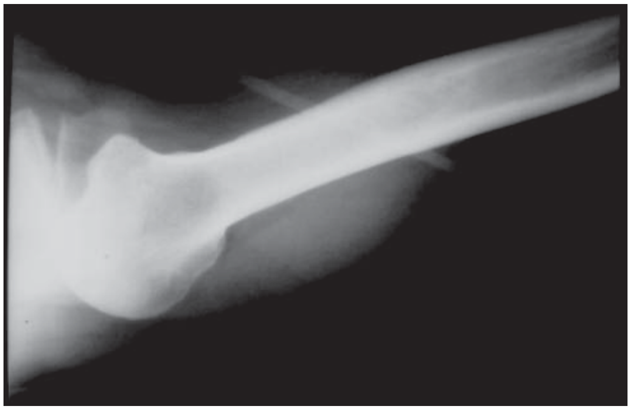


Fig. 9-56. Axillary view of posterior dislocation of the humeral head, which may be associated with injuries of the axillary nerve and posterior cord of the brachial plexus. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center. Washington, DC.

corresponding sensory NCSs are normal. In postganglionic nerve insult, however, pertinent SNAPs are either significantly reduced in amplitude or absent. This phenomenon occurs as a result of proximal axonal injury with resulting axonal degeneration distal to the site of insult.

It is important to recognize that SNAP amplitudes will not be abnormal for at least the first 3 to 5 days following proximal postganglionic nerve insult and may continue to conduct impulses for up to 2 weeks. It is also significant that temporal dispersion of the SNAP is not characteristic of most plexopathies, assuming there is not a concomitant systemic disorder or underlying polyneuropathy or both, or an isolated peripheral entrapment neuropathy. Sensory NCSs may provide valuable clues regarding the presence of a postganglionic brachial plexopathy, but are limited by the fact that sensory nerve studies may not be available for the suspected level of brachial plexus pathology. In addition, it is recognized that needle electrode evaluation is more sensitive in detecting and localizing nerve pathology than sensory NCSs.

Axonotmesis appears to be the most frequent pathological process associated with plexopathies.[405] Conduction abnormalities occurring in plexopathies include proximal focal slowing or conduction block, or both. Distal motor and sensory conduction velocities are not usually significantly affected in the presence of incomplete proximal injury, although slightly decreased conduction velocities may be recorded.

Thoracic Outlet Syndrome

Anatomic Considerations

The thoracic outlet syndrome (TOS) typically involves the lower trunk of the brachial plexus and may be secondary to neurologic or vascular disturbance, or both. It may, on occasion, affect the middle trunk as well. It has been described in various terms based on the proposed site and mechanism of presumed compression injury. These include compression secondary to cervical rib anomaly[406] and scalene anticus,[407] costoclavicular,[408] and hyperabduction syndromes.[409]

Three classifications of TOS are currently recognized[410]:

1. True, classic, or neurogenic TOS with objective neurologic impairment.
2. Disputed or symptomatic TOS with evanescent neurogenic symptoms and normal physical exam.
3. TOS secondary to vascular compression.

True neurogenic TOS is rare.[411] Neurologic impairment as a result of lower brachial plexus injury is felt to occur potentially at several sites[9]:

- posterior border of Sibson's fascia;
- crescentic tendinous fibers of the scalenus medius and scalenus anterior muscle;
- between the narrow tendinous angle between the scalenus medius and scalenus minimis, or between the scalenus medius and scalenus anterior muscles;
- sharp tendinous posterior border of the scalenus anterior muscle;
- abnormal rib or ligamentous extensions associated with the rib;
- bony prominence on the first rib;
- between the clavicle and a normal or abnormal rib; and
- between the aneurysm of the subclavian artery and underlying structures.

Etiology

The fundamental cause of TOS appears to be due to intermittent or chronic compression injury. This may occur from malalignment of normal structures, or may be secondary to an anomalous fibrous band. This band may attach to a cervical rib, but has also been noted to attach to the first thoracic rib or other osseous structures. The anterior scalene syndrome is felt to result from lower plexus compression injury between the anterior and medial scalene muscles. This appears to be a particular problem for competitive swimmers in whom both scalene muscle hypertrophy and repetitive arm action predispose the athlete to plexus injury.[412] The costoclavicular syndrome implies lower plexus injury secondary to compression injury by the ribs or clavicle or both. An additional "subsyndrome" is the pectoralis minor syndrome, which is defined as plexus injury as it enters the axilla between the upper ribs and the pectoralis minor muscle. This subset of TOS is also felt to be precipitated or exacerbated by repetitive arm activity in the presence of pectoralis minor hypertrophy.[412]

Clinical Presentation

The actual frequency of TOS is unknown. Early reports of TOS may not have been lower trunk compression injury but CTS or cervical radiculopathy instead.[9,413]

The signs and symptoms of TOS are often vague. Physical examination findings are typically poorly defined, but it should be in the differential diagnosis in patients presenting with obscure upper extremity pain and numbness, along with weakness and atrophy of hand intrinsic muscles (Table 9-14).

Musicians, particularly flutists and violinists, are felt to be at particular risk for developing TOS.[414,415] There is a significantly greater incidence of TOS in females. The ratio is 9:1 for neurogenic TOS. Posture may play a role in this female predilection since drooping shoulders are more common in women.[410,416] True neurogenic TOS often initially presents with paresthesias, which may be followed by persistent pain along with development of weakness and muscle atrophy. The distribution of signs and symptoms generally follow the distribution of the medial cutaneous nerve of the forearm and the ulnar nerve. Pain is described as a poorly localized aching discomfort but may at times affect the whole arm. The pain is usually exacerbated with repetitive or heavy lifting or prolonged playing of selected musical instruments. There is usually an accompanying sensory deficit in the areas innervated by the medial cutaneous nerve of the forearm and the ulnar nerve.

The physical exam in TOS may offer few objective clues. The Adson maneuver, consisting of ipsilateral shoulder abduction and external rotation along with scapular retraction and neck rotation while monitoring for a decrease in the radial pulse, is felt to be an unreliable test, especially for neurogenic TOS.[412]

TABLE 9-14

MASQUERADERS OF THORACIC OUTLET SYNDROME

Pathology	Conditions
Neurologic	Multiple Sclerosis
	Parsonage Turner syndrome
	Traumatic brachial plexopathy
	Brachial plexopathy associated with Pancoast tumor
	Carpal tunnel syndrome
	Ulnar neuropathy
	Complex region pain syndrome
Intramedullary	Syringomyelia
	Spinal cord infarction
	Gliomas
Extramedullary	Neurofibromas
	Meningiomas
	Cervical spondylosis
	Herniated cervical intervertebral disk
Vascular	Atherosclerosis
	Systemic lupus erythematosus
	Aortic dissection

A variety of conditions and pressure from various soft tissue masses potentially mimic TOS and may need to be excluded. These include intramedullary and extramedullary disease processes as well as pathologies which may cause vascular insufficiency (see Table 9-14).

Electrodiagnosis

Nerve conduction studies have limited usefulness in identifying TOS. They are most helpful in excluding other conditions, particularly CTS and ulnar neuropathy. Sensitive nerve conduction findings supportive of a diagnosis of TOS include decreased ulnar SNAP amplitude at the wrist and decreased mixed nerve potential amplitude at the elbow. This drop in amplitude is attributable to axonal injury specifically affecting nerves at the level of the brachial plexus, which are destined to emerge as ulnar sensory fibers.[417] Other studies[417,418] have indicated that patients with TOS may show decreased median CMAP along with a decreased ulnar SNAP amplitude. The latter nerve conduction findings are currently felt to be the most sensitive criteria for neurologic TOS.

Proximal NCSs from Erb's point to an ulnar innervated muscle may have some merit if delayed conduction can be documented.[413,419] Some authors,[420,421] however, have not documented any abnormality with NCSs. It is not always clear whether the actual lesion is below, at, or above this site of stimulation. Furthermore, supramaximal stimulation is required that may result in stimulation of the nerve distal to the intended site, and there is significant potential for error when measuring from site of stimulation to the active electrode.

C-8 nerve root simulation, which has the advantage of definite stimulation proximal to Erb's point, has been advocated.[412] However, this technique is primarily useful in identifying focal demyelination injury, which is not characteristic of TOS. Nerve root stimulation is not currently an established technique for confirming TOS.[421] Weber and Kahn suggest that ulnar F-waves may be an effective way of studying conduction across the thoracic outlet.[412] Dawson and colleagues[422] acknowledge that there may be prolonged F-wave latencies in unambiguous TOS, but feel this slowing is not a sensitive measurement for this syndrome. Electromyographic studies may be helpful in confirming the presence of chronic denervation activity in the thenar and hypothenar muscles.

Treatment

A number of nonsurgical measures have been advocated to treat TOS. Arm restraints to prevent excessive shoulder abduction during sleep[422] and selective upper body exercises[423] have been suggested. A number of authors[424–426] have documented significant success with a formal exercise program.

Sunderland[9] has identified specific criteria for determining failure of conservative management. These include signs of muscle wasting, intermittent paresthesia followed by sensory loss, and progressive pain to the point of incapacitation.

Surgical management of TOS most commonly involves first cervical rib resection via the transaxillary approach, but this procedure remains controversial and indications are quite variable and not well-established. When constricting bands are identified during the procedure, they are released. Supraclavicular exploration is the procedure of choice for some surgeons.[413,427] It has the advantage of visualizing a cervical rib directly within the field and if constricting bands are present, they may be dissected without the need to remove the first rib. However, its disadvantages are less acceptable cosmesis, potential phrenic or long thoracic nerve injury or both, and more extensive dissection.[422] Unfortunately, severe, persistent CRPS-Type II and functional deficit can result from surgical intervention. Thus, it is prudent to limit surgical management to those patients with unequivocal clinical evidence of true neurogenic or true vascular compromise.[422]

Radial Nerve Injuries and Compression Syndromes

The radial nerve is especially vulnerable to injury at several sites. These include injury to the main trunk of the radial nerve at the level of the spiral groove, posterior interosseous injury at the elbow, and entrapment or injury of the superficial radial nerve in the distal third portion of the forearm or at the wrist.

Injuries and Compression in the Spiral Groove Area

Anatomical considerations. The radial nerve is a branch of the posterior cord of the brachial plexus with neural fiber contributions from the fifth to eighth cervical and first thoracic nerve roots (Figure 9-57). It begins at the lower border of the pectoralis minor. In the arm, it winds around the posterior aspect of the humerus and passes along the musculospiral groove. It emerges anterior to the distal arm about proximal to the lateral epicondyle. In the upper arm, the radial nerve branches innervate the three heads of the triceps, the anconeus, and the upper portion of the forearm extensors and supinator muscles. Sensory branches of the radial nerve include the posterior brachial cutaneous, posterior antebrachial cutaneous, and superficial radial nerves. The posterior aspect of the upper arm is supplied by the posterior brachial cutaneous nerve. The dorsal surface of the forearm is supplied by the posterior antebrachial cutaneous nerve. The superficial radial nerve provides sensory innervation to the dorsal aspect of the radial half of the hand.

Radial nerve branches to the long and medial heads of the triceps arise in the axilla. The radial nerve may be accompanied in the spiral groove by the branches to the medial and lateral heads of the triceps. The main radial nerve trunk typically lies directly against the humerus in the groove area, but its branches lie in a plane between the heads of the triceps. Thus, the main radial nerve bundle is more

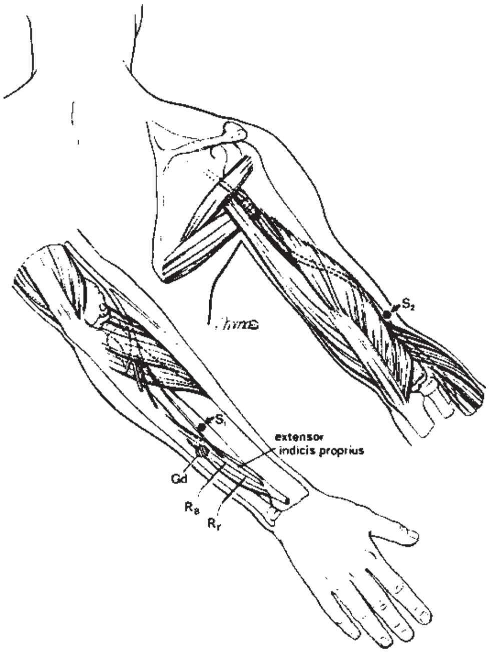


Fig. 9-57. The radial nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 73.

vulnerable to injury secondary to mid-third fractures of the humeral shaft. This is in contrast to crutch palsy in which both the radial nerve branches and the main radial nerve suffer compression injury between a crutch and the underlying muscle tissue.[9]

The posterior cutaneous nerve of the arm and forearm accompanies the radial nerve to the spiral groove but lies more posterior relative to the humeral shaft. Thus, it too is less vulnerable to injury than the main radial nerve in the event of mid-third humeral fractures.[9]

As the radial nerve emerges from the spiral groove, it sends a branch to the brachioradialis, followed slightly more distally by a branch to the extensor carpi radialis longus. It then passes between the brachialis and brachioradialis before entering the forearm.

Etiology. The radial nerve may incur injury as a result of direct, prolonged pressure, such as in Saturday night palsy.[428] This form of nerve lesion characteristically occurs in cachectic and fatigued persons who are intoxicated or suffering from drug narcosis. Thus, they are in a state in which they fail to perceive and react to the stages of progressive nerve injury.

Humeral fractures, as previously noted, may be associated with radial nerve injuries in the upper arm.[429,430] Injury may occur as a result of direct injury associated with the trauma causing the fracture or by the fracture edges. Also, the radial nerve is relatively fixed where it leaves the spiral groove. This predisposes it to traction injury when the fractured segments of the humerus are traumatically separated.[9] Patients sustaining fractures of the distal third of the humerus are also at risk for radial nerve injury.

Radial nerve entrapment has been documented to occur in an area of drug-induced (pentazocine) fibrotic changes within the lateral head of the triceps.[431] Radial nerve injury has also been attributed to compression by this muscle during severe muscular exertion.[432] Lotem et al[433] reported three cases of radial nerve injury manifested by transient paresis following intense muscular effort, specifically elbow extension. Sunderland cites Gowers in 1892 who wrote,

> I have three times seen paralysis from a violent contraction of the triceps, once during the act of pulling on a tight pair of boots, once from throwing a stone with energy, and once from grasping a lamp-post to avoid a fall during a severe attack of giddiness. In each the nerve was at once completely paralyzed; and in the second, in which the palsy was severe, a bruised appearance was observed over the lower part of the triceps.[9]

Clinical presentation. Radial nerve compression at the spiral groove may cause wrist drop due to wrist and finger extension weakness, along with weakness of the brachioradialis. The triceps muscles are typically spared. Sensory impairment may involve the dorsal interspace between the thumb and index finger and/or the proximal halves of the thumb, index finger, and middle finger.[389]

Treatment. Treatment is directed at surgically removing the offending compressive mass or eliminating the precipitating posture. Wrist-hand splints with the hand and wrist placed in a position of function may be prescribed (Figure 9-58). Any splinting should be coupled with a daily range-of-motion exercise program, both passive and active assistive depending on the degree of weakness present.

Posterior Interosseous Nerve Entrapment

Anatomic considerations. The posterior interosseous nerve is a motor branch of the radial nerve (Figure 9-59). The radial nerve bifurcates into

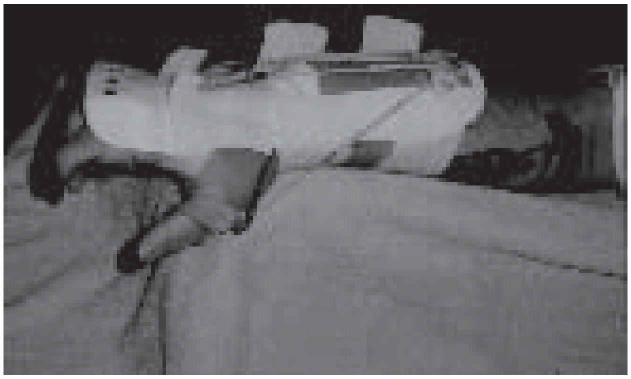


Fig. 9-58. Dynamic dorsal wrist hand orthosis with low profile outriggers assisting metacarpophalangeal extension and thumb abduction. The wrist is set in a functional position.

a sensory branch and the posterior interosseous nerve, usually at the level of the radiocapitellar joint. In some cases, it may separate 2-5 cm proximal or distal to this joint.[434] When the nerve is directly anterior to the radiohumeral joint capsule and the radial head, it lies lateral to the biceps tendon and bicipital bursa and medial to the supinator muscle.[9] It then passes between the two heads of the supinator muscle and innervates this muscle as it courses through it. It proceeds dorsolaterally around the neck of the radius within the substance of the supinator muscle. The extensor carpi radialis brevis is innervated by the radial nerve at or distal to the radial head.

The posterior interosseous nerve subsequently separates into two groups comprised of multiple branches. One group innervates the superficially lying forearm extensor muscles, including the extensor digitorum communis, extensor digiti quinti, and extensor carpi ulnaris. The other innervates the deeper lying forearm extensors, including the abductor pollicis longus, extensor pollicis longus and brevis, and the extensor indicis proprius.

The posterior interosseous nerve enters the supinator through an inverted, fibrous arch known as the arcade of Frohse. It is formed by the thickened tendinous edge of the proximal border of the superficial head of the supinator.

Etiology. The posterior interosseous nerve is susceptible to both intrinsic and extrinsic compression injury. It can be entrapped by the aforementioned arcade of Frohse,[435–437] the supinator,[438] tumors (most commonly lipomas),[439,440] ganglia, and rarely, elbow synovitis.[422,441] It has also been documented to occur after fracture or dislocation of the radial head and specifically with Monteggia fractures (radial head dislocation along with fracture of the ulna)[442–444] and as a complication of Canadian forearm crutch use.[445] In some of patients, the posterior interosseous nerve is said to lie directly on the periosteum of the proximal radius, making it particularly susceptible to damage by a fracture of the proximal radius or by a proximally placed metal plate used to stabilize the fracture.[434] Vascular leashes enveloping the nerve have been found to be potential causes of posterior interosseous nerve compression.[422]

Clinical presentation. Patients present with compromised wrist extension strength. They also have finger extension weakness at the MCP joints and thumb extension and abduction paresis. They retain radially deviated wrist extension because the extensor carpi radialis longus and brevis muscles are spared, each receiving a branch from the radial nerve proximal to the supinator. The brachialis and triceps are also spared. Prior to developing the combination of wrist, thumb, and finger extension weakness, these patients may present with isolated inability to fully extend their little and ring fingers.

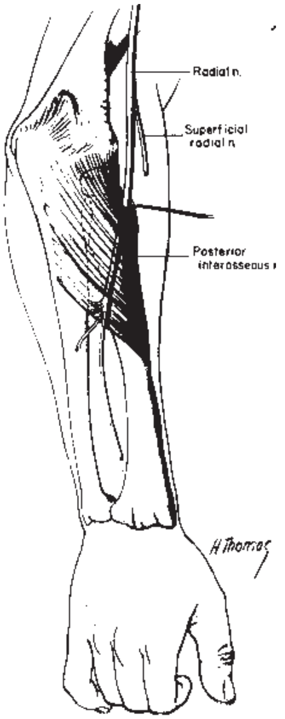


Fig. 9-59. The posterior interosseous branch of the radial nerve. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 35.

Muscles that are spared following a posterior interosseous compression neuropathy include extensor carpi radialis longus, the extensor carpi radialis brevis, and the supinator. The muscles that are affected are the extensor carpi ulnaris, including the extensor digitorum communis, extensor indicis proprius, abductor pollicis longus, extensor pollicis brevis, and the extensor pollicis longus.

Although the posterior interosseous nerve is a motor nerve, compression injury has caused paresthesias in some cases.[446] Pain begins at the elbow or proximal forearm area in about half of the patients.[447] Pain is usually acute, although it may also be gradual in onset. There may be reproducible tenderness with palpation over the forearm extensor muscle mass just distal to the radial head.

Electrodiagnosis. Nerve conduction studies of the radial nerve in the forearm have shown a prolonged motor latency consistent with focal slowing involving the posterior interosseous nerve. The study is performed by stimulating the radial nerve at the level of the elbow with active electrode pickup of the action potential on the extensor digitorum communis muscle.[448] The superficial radial NCS is normal. Electromyography may reveal spontaneous activity consistent with muscle membrane instability and suggestive of denervation activity involving all radially innervated forearm muscles except for the extensor carpi radialis longus and brevis and supinator muscles.

Treatment. In most cases, clinically or electrodiagnostically or both, documented posterior interosseous nerve entrapment is managed surgically. This includes exploration and resection of the offending mass, such as a tumor or ganglia, or the release of compressive fibrous bands.

A wrist-hand splint fabricated in a position of function may be used pre- or postoperatively to address the incomplete wrist weakness and finger and thumb weakness. A static splint may be used at rest and a low-profile dynamic extension splint used during the day. Daily passive and active wrist flexion and extension to the degree capable should also be included in the therapeutic program.

Superficial Radial Nerve Compression

The superficial radial nerve courses subcutaneously along the lateral aspect of the radius. It is particularly vulnerable to extrinsic pressure because of its superficial position. Excessively tight wristbands or handcuffs may cause focal nerve compression injury resulting in sensory impairment involving the radial aspect of the hand. This nerve may take an aberrant course or there may be variations in the distribution of cutaneous innervation, thus confusing the clinical picture.

There are multiple documented causes of superficial radial nerve compression injury[412,434,449]: constricting wrist bands, handcuffs, forearm hemorrhage, excessively tight surgical gloves, vein cutdown procedure, operative repair of deQuervain's tenosynovitis, casting for Colle's fracture, schwannoma of the superficial radial nerve, and fibrous tendon joining brachioradialis and extensor carpi radialis.

Injury to the superficial radial nerve results in isolated numbness, paresthesias, or painful dysesthesia without motor deficits. There may be a positive Tinel's sign in the lateral forearm area. Symptoms may be enhanced with placing the forearm in end-range pronation coupled with ulnar wrist flexion.[450]

Nerve conduction studies of the superficial radial nerve may result in an absent response, or prolonged latency with attenuation of the amplitude and temporal dispersion of the sensory action potential. Electromyography is of no value in this syndrome except to exclude other nerve pathology.

Treatment is directed at removing the offending constriction or focal pressure. Causalgias related to superficial nerve compression have been documented as a complication of some superficial radial nerve lesions.[451] Nerve repair, excision of an associated neuroma, and cervical sympathectomies have been performed to address this problem with some success.[434]

Median Nerve Injuries and Compression Syndromes

The median nerve is vulnerable to compression injury at three major sites. At the elbow; at the ligament of Struthers, where the main trunk of the median nerve may be entrapped; or between the two heads of the pronator teres. In the forearm, the anterior interosseous nerve, a motor branch of the median nerve, is subject to compression. The most common entrapment neuropathy, CTS, occurs secondary to compression at the distal edge of the transverse carpal ligament or, less commonly at the intermetacarpal tunnel.[160] A number of less common sites of median nerve compression have been observed[411]:

- post stupor with proximal median nerve compression at entrance to canalis brachialis at inferior border of pectoralis major;

- injury secondary to fracture of distal third of humerus;
- entrapment by lacertus fibrosis (fibrous band connecting biceps tendon to flexor carpi radialis);
- injury secondary to elbow dislocation;
- injury secondary to fracture—dislocation of distal radioulnar joint;
- entrapment by hemodialysis loop grafts at both the elbow and in the forearm;
- entrapment by lateral border of flexor digitorum superficialis;
- distal forearm fibrovascular band;
- radial artery puncture at the wrist with direct trauma or injury secondary to bleeding; and
- compression injury of palmar cutaneous branch secondary to ganglia.

Supracondylar Process Syndrome

Anatomic considerations. The ligament of Struthers is an anomalous fibrous band (Figure 9-60). It has been documented to have an incidence of 0.7% to 2.7%.[452] It extends from its attachment on the supracondylar process of the distal anteromedial humerus (usually via a bony spur) to the medial epicondyle.[160] The bony spur may be palpable and is evident on a radiograph.[453] The ligament may be bilateral[411] and is not always associated with a bony spur.[454]

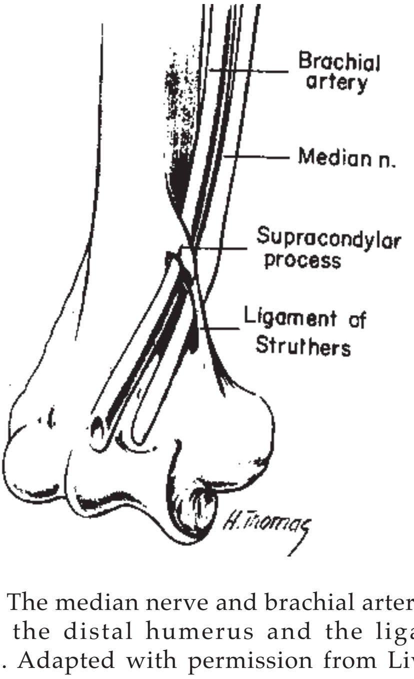


Fig. 9-60. The median nerve and brachial artery passing between the distal humerus and the ligament of Struthers. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 28.

Etiology. The median nerve may be entrapped under the ligament of Struthers just proximal to the antecubital space. Some feel the compression injury is due to a combination of static impingement and dynamic injury associated with repetitive motion at the elbow.[412]

Clinical presentation. This syndrome may result in weakness of all median nerve innervated forearm and hand muscles, including the pronator teres. It may also result in sensory deficits in the hand in the distribution of the median nerve. The brachial artery also passes under the ligament of Struthers when it is present. If it too is compressed, a patient may present with distal vascular and neuropathic changes.[411]

Electrodiagnosis. Median nerve conduction velocity across the site of presumed entrapment at the elbow may be slowed.[455,456] An EMG of selected forearm and hand muscles innervated by the median nerve may show evidence of denervation activity.

Pronator (Teres) Syndrome

Anatomic considerations. The median nerve crosses the elbow in close approximation to the brachial artery and biceps tendon. The biceps tendon is lateral, the median nerve is medial, and the brachial artery courses between these two structures. The median nerve enters the forearm and passes under the lacertus fibrosus, a thick fascial band extending from the biceps tendon to the forearm fascia. It then courses between the superficial and deep head of the pronator teres and passes beneath the fibrous arch of the flexor digitorum superficialis.

Etiology. The median nerve may be compressed by a thickened lacertus fibrosus, by a hypertrophied pronator teres or fibrous band within this muscle, between the superficial and deep heads of the pronator teres, or by the edge of the fibrous arch of the flexor digitorum superficialis (Figure 9-61).[422] Fractures of the forearm as well as elbow dislocation may secondarily injure the median nerve (Figure 9-62). Median nerve injury in this forearm area has also been documented in association with compartment syndrome.[457] A persistent median artery as a cause of pronator teres syndrome, secondary to compression of the nerve by the artery, was reported by Jones and Ming.[458] Luce et al[459] noted that in patients on anticoagulants, repeated attempts at brachial artery puncture may also cause median nerve injury. This compressive injury secondary to bleeding may develop insidiously.

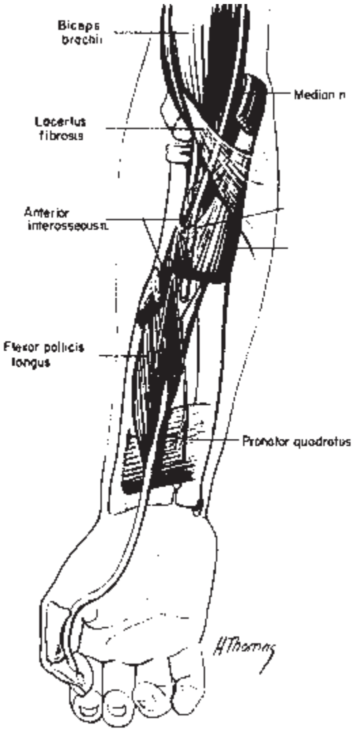


Fig. 9-61. The median nerve may be entrapped by the lacertus fibrosis or the fibrous arch of the flexor digitorum sublimis (superficialis) as well as between the deep and superficial heads of the pronator teres muscle. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 25.

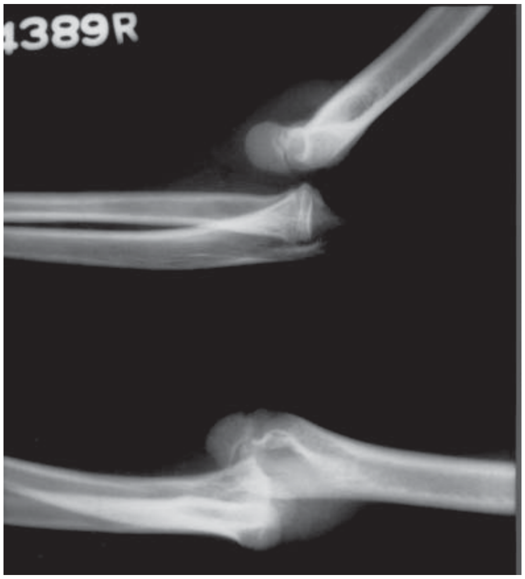


Fig. 9-62. The median nerve may be injured following dislocation of the elbow. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

Clinical presentation. Patients usually complain of an aching discomfort in the proximal forearm, exacerbated by activities requiring repetitive or strenuous forearm pronation. There may be proximal radiation to the elbow or even the shoulder, along with a sense of heaviness and easy fatigability with use of the arm. Poorly localized paresthesias may be present, usually intermittently if at all. Nocturnal exacerbation of symptoms is not characteristic.

Physical examination usually reveals dull tenderness with palpation over the pronator teres muscle mass. The pronator teres may appear enlarged upon palpation. Occasionally, a sharp pain will be elicited instead, and a tap over the pronator teres may produce a Tinel's sign.

Muscle strength testing reveals variable weakness of the median nerve innervated forearm flexors, including the palmaris longus, flexor carpi radialis, flexor digitorum profundus (radial portion), flexor digitorum superficialis, flexor pollicis longus, and pronator quadratus. The pronator teres, on the other hand, is typically spared because branches to this muscle emerge from the median nerve prior to the aforementioned entrapment sites. There is also variable weakness of the median innervated hand intrinsics.

Spinner describes three tests that may be helpful in clinically supporting the presence of pronator teres syndrome.[434]

1. One test is performed by placing the forearm in full pronation and the wrist in flexion. An examiner then passively supinates the forearm and extends the wrist while the patient actively resists this maneuver. A positive test is manifested by precipitation or exacerbation of pain in the proximal forearm and suggests median nerve compression in this area by the pronator teres.
2. A second test involves full supination of the forearm concomitantly with elbow flexion. The examiner passively pronates the forearm while the patient resists the action. The patient's active biceps contraction causes tightening of the lacertus fibrosus. A positive test is manifested by precipitation or exaggeration of symptoms and suggests that the lacertus fibrosus is the compressive structure.
3. A third test directs the patient to flex the proximal IP joint of the middle finger against resistance, thus activating the flexor digitorum sublimis muscle. If this

> test precipitates or increases the patient's discomfort, it is suspected that the arch of the flexor digitorum sublimis may be causing median compression injury.

Electrodiagnosis. The pronator teres syndrome is primarily a clinical diagnosis. Electrodiagnostic testing detects abnormalities in only a minority of patients studied.[422] Median motor and sensory conduction velocities may be slowed in the forearm segment. The sensory action potential amplitude distally may be reduced with compression injury of the median nerve in the forearm. Nerve conduction studies are infrequently abnormal, but can be helpful in excluding other neuropathic conditions in the differential, including CTS. Electromyography provides the most useful information when abnormal findings, which are suggestive of denervation activity, are found in both median-innervated forearm and hand intrinsic muscles, excluding the pronator teres.[454]

Treatment. Conservative management includes avoidance of precipitating or exacerbating activity and use of NSAIDs. Temporary use of static elbow and wrist splints in a position of rest of the biceps, supinator, pronator teres, and forearm wrist flexors, followed by gently progressive range-of-motion activities to restore pain-free mobility, may be helpful in some patients.

In patients who sustain acute median nerve compression in the proximal forearm secondary to crush injury, bleeding, or other causes of increased intracompartmental pressure, fasciotomy with median nerve decompression is advocated. If this surgical intervention is performed in a timely fashion, there is excellent prognosis for median nerve recovery. This may not be the case for individuals suffering from chronic median nerve compression. Their prognosis for recovery is guarded.[422]

Anterior Interosseous Syndrome

Anatomic considerations. The anterior interosseous branches off the main median nerve approximately 5 to 8 cm distal to the lateral epicondyle. It is predominantly a motor nerve. Although it has no superficial sensory fibers, it does carry pain and proprioception fibers from deep forearm soft tissue structures and from the wrist joints, including the radiocarpal, intercarpal and carpometacarpal, and distal radioulnar joints.[422] It innervates three forearm muscles, the flexor pollicis longus, the flexor digitorum profundus (radial portion), and the pronator quadratus.

Etiology. The anterior interosseous syndrome is also known as the syndrome of Kiloh and Nevin.[460] The site of injury is just distal to the pronator teres muscle. The anterior interosseous nerve may be selectively compressed by any number of structures or directly injured secondary to trauma.

Causes of anterior interosseous syndrome[411,422] include

- tendinous origin of deep head of pronator teres;
- tendinous origin of flexor digitorum sublimis to the middle finger;
- accessory muscles and tendons from the flexor digitorum, flexor pronator quadratus, and other forearm muscles;
- fibrous bands of the flexor digitorum sublimis or flexor digitorum profundus;
- aberrant vessels and thrombosed collateral vessels;
- excessive forearm exertion;
- direct trauma;
- forearm fractures, lacerations, gunshot wounds;
- drug injections;
- postoperative complication of open reduction internal fixation of forearm fractures;
- venus cutdowns; and
- extrinsic pressure (cast, heavy handbag, prolonged leaning on forearm).

Collins and Weber[461] describe actual avulsion of the anterior interosseous nerve secondary to trauma. Anterior interosseous syndrome has also been documented to occur in association with metastatic bronchiogenic carcinoma involvement of the forearm.[462]

The anterior interosseous syndrome may be clinically mimicked by more proximal inflammatory nerve lesions or more proximal compression injury. For example, the anterior interosseous neural fibers can be selectively involved in brachial neuritis (neuralgic amyotrophy or Parsonage-Turner syndrome).[463,464] A pseudo-anterior interosseous nerve syndrome involves partial median nerve injury secondary to catheterization at the antecubital fossa level, presumably selectively involving nerve fibers specifically destined to form the anterior interosseous nerve. Elbow dislocations have been documented to produce a similar presentation (see Figure 9-62).[465,466]

Rupture or entrapment of the flexor pollicis longus tendon will cause isolated inability to flex the IP joint of the thumb. This tendon, along with the flexor digitorum profundus to the index finger, is especially vulnerable to rupture in rheumatoid arthritis on the sharp edge of an eroded scaphoid tubercle. This phenomenon can mimic a partial anterior interosseous syndrome. A history of locking or snapping of the thumb at this joint and the absence of any weakness of the pronator quadratus or flexor digitorum profundus muscle to the middle finger will help to differentiate this problem from the anterior interosseous syndrome. If due to rheumatoid arthritis, one may detect clinical evidence of prominent flexor tenosynovitis, and wrist radiographs may reveal scaphoid bone erosion.[422]

Clinical presentation. Patients classically present with acute pain in the proximal forearm region and sometimes in the elbow area as well. They do not typically have any sensory symptoms, although poorly localized and described sensory complaints may be noted on occasion. Weakness is variable and limited to the flexor pollicis longus, flexor digitorum profundus to the index and middle fingers, and the pronator quadratus. These patients experience difficulty making the circular "OK" sign with their thumb and index finger. They form a triangle instead because of the weakness at the IP joint of the thumb and the distal IP joint of the index finger. For the same reason, they are unable to pinch with the tips of their thumb and index finger effectively. They typically have variable loss of dexterity in fine motor tasks as well.

Electrodiagnosis. Conventional median motor NCSs, which involve stimulation at the wrist and from above the elbow with pickup at the abductor pollicis brevis muscle, are normal. However, median motor conduction from the elbow to pickup over the pronator quadratus may reveal a prolonged latency and temporal dispersion of the CMAP.[467] Median sensory NCS is normal and its value is primarily the exclusion of other median nerve pathology. Elecromyography may reveal evidence of variable degrees of denervation activity involving the flexor digitorum profundus I and II, flexor pollicis longus, and pronator quadratus.

Treatment. Management of anterior interosseous nerve injury is highly dependent on the cause. If a penetrating wound is the cause, immediate surgical exploration and repair is warranted. If a crush injury with impending Volksmann ischemic contracture is the cause, immediate decompression is clearly indicated. If it is spontaneous or clearly related to a given activity, relative rest with avoidance of the precipitating or exacerbating activity may be sufficient to provide relief. Temporary splinting of the forearm may offer greater assurance of adequate immobilization and rest. Nonsteroidal antiinflammatory medications and local steroid injections in the region of the pronator teres may be helpful in alleviating persistent discomfort.

There is little consensus regarding timing of surgical intervention in patients who suffer persistent discomfort despite conservative management. However, the surgical literature tends to favor surgical exploration in those patients with spontaneous onset of a complete anterior interosseous nerve deficit if they show no significant improvement after 12 weeks.[468,469] Most favor waiting an even longer period of time in patients who present with only incomplete anterior interosseous nerve palsy.

Dawson and colleagues[422] suggest a period of at least 6 months of conservative treatment in patients with spontaneous anterior interosseous syndrome. This rationale is based on their experience that most cases of spontaneous anterior interosseous nerve palsy are due to a neuritis rather than compression injury and that these patients characteristically recover completely within about ten months after onset.[470]

Carpal Tunnel Syndrome

Anatomic considerations. Prior to entering the carpal tunnel, the palmar cutaneous nerve is the last major branch of the median nerve in the forearm, usually arising about 3 to 4 cm proximal to the proximal edge of the transverse carpal ligament. Thus, it is spared in CTS. It serves the thenar region of the hand and a variable portion of the palm.

The median nerve becomes more superficial at the distal forearm between the tendons of the palmaris longus and the flexor carpi radialis. It then passes through the fibroosseous carpal tunnel (Figure 9-63). The radial wall of this tunnel is formed by the scaphoid and trapezium and ulnar wall by the pisiform and hamate. The lunate, capitate, trapezoid, and associated ligamentous structures form its floor or dorsal surface. The transverse carpal forms the roof or volar surface. The transverse carpal ligament attaches to the tubercle of the scaphoid and trapezium laterally and the hamate medially. Nine extrinsic wrist flexor tendons accompany the median nerve through this tunnel. They include the flexor pollicis longus (1), flexor digitorum superficialis (4), and flexor digitorum profundus (4) tendons. The distal volar skin crease of the wrist corresponds to the proximal border of the carpal tunnel, which extends 3 cm distal to this crease.[422,434] The carpal tunnel narrows in cross-section at 2 to 2.5 cm distal to the entrance

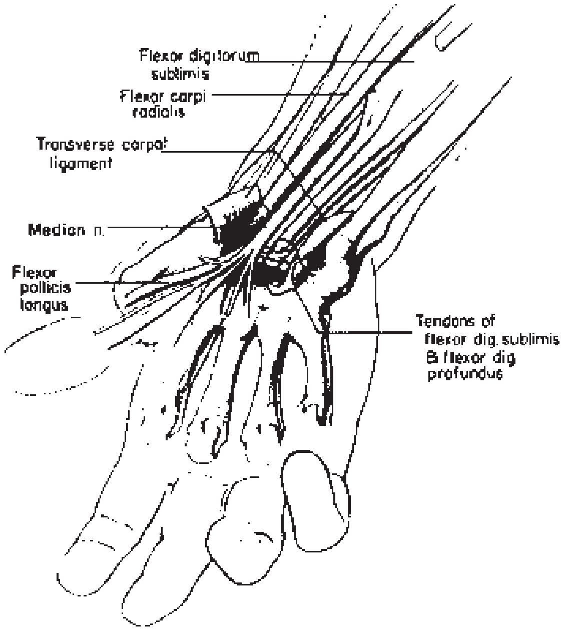

Flexor digitorum sublimis
Flexor carpi radialis
Transverse carpal ligament
Median n.
Flexor pollicis longus
Tendons of flexor dig. sublimis & flexor dig profundus

Fig. 9-63. The carpal tunnel. The median nerve, tendons of both the flexor digitorum profundus and superficialis, and flexor pollicis longus travel beneath the transverse carpal ligament which has been reflected. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 20.

of the tunnel.[471] In CTS, the size of the myelinated nerve fibers are significantly reduced at this point.[472]

As the median nerve emerges from the carpal tunnel, it splays outward into both motor and sensory fibers. The motor nerve, in close proximity to the common palmar digital nerve to the thumb, curves over or through the flexor pollicis brevis. It supplies the superficial head of the flexor pollicis brevis as it courses over or through it and subsequently divides to serve the other thenar muscles, the abductor pollicis brevis and the opponens pollicis.

The sensory fibers classically serve the skin of the radial aspect of the palm and volar surface of the thumb, index, and middle fingers, and the radial half of the ring finger. Dorsally, sensory fibers from the median nerve supply the skin distal to the proximal IP joints of the index and middle fingers and the radial aspect of the little finger. The dorsal surface of the distal thumb may be innervated by either the median or radial nerve.[422,473] It is clinically important to be aware that a multitude of anatomic variations exist regarding median sensory supply to the hand and digits.

Etiology**.** Carpal tunnel syndrome is the most common entrapment neuropathy. Space-occupying lesions within the carpal tunnel are the most common cause of this syndrome. A wide variety of conditions are associated with CTS. All of them, to some degree, compromise the limited space within this relatively unyielding fibro-osseous structure.

Acute CTS occurs as a complication of multiple traumatic injuries involving the wrist or hand. It has been associated with Colle's fracture (Figure 9-64).[474] It has also been documented to occur after epiphyseal fracture of the distal radius, after fracture of both the ulna and radial bones, and after dislocation or fracture-dislocation of the carpus.[475,476] Suppurative flexor tenosynovitis has been documented to cause abrupt onset of CTS as well. It may be complicated by acute median nerve paralysis requiring immediate decompression to prevent irreparable damage.[422]

Crush injury of the hand or wrist results in marked swelling and may be complicated by Volkmann's ischemic contracture. Such patients are at high risk for developing acute CTS. Individuals taking anticoagulants are at increased risk for bleeding into the carpal tunnel[477] or into the median nerve itself,[478] with consequent development of acute or chronic CTS.

Chronic CTS may occur as result of any of the aforementioned traumatic injuries. It may also develop secondary to prolonged or repetitive extrinsic trauma, such as occasionally occurs after walking with axillary crutches. Crutch walking in a susceptible patient has been documented to cause

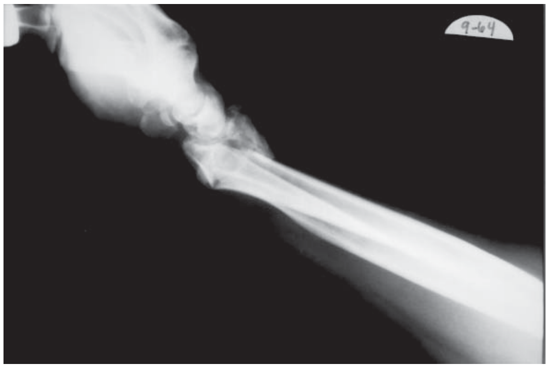


Fig. 9-64. Distal radial fracture with dorsally deviated distal segment (Colle's fracture), which may be associated with median nerve entrapment at the wrist. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

chronic compression injury of the motor branch of the median nerve.[422]

Clinical presentation. Carpal tunnel syndrome is a consideration in any patient who presents with numbness, painful dysesthesias, weakness, and impaired dexterity of the hand. In most patients, there is a characteristic presentation, but a significant number present in an atypical fashion and require electrophysiologic testing for definitive diagnosis. Carpal tunnel syndrome affects females more often than males. The most common age of onset is in the fifth or sixth decades, although many present earlier, particularly if their daily activities or occupation involves extensive manual labor. Symptoms most often affect the dominant hand,[479] but bilateral complaints are not uncommon.

Numbness classically involves the thumb, index finger, and radial half of the ring finger. Some have found that numbness is most commonly present in middle finger or both the middle and index fingers.[422] Pain often occurs at the wrist with proximal radiation but may be present in the hand, forearm, elbow, and shoulder as well. The pain is described as an aching, cramping, or burning discomfort. Shaking or rubbing the hand may offer momentary relief. Weakness is not a typical complaint, although patients may complain of tiredness and easy fatigability when performing manual activities. They may also complain of dropping items with increasing frequency. However, this is most likely related to their sensory deficit rather than weakness. There may be an increased sensitivity to cold, and in some individuals with advanced disease, vasotrophic changes, including excessive sweating, edema, and skin color changes, may occur.

Early in the course of CTS, there are generally no objective physical findings. Later, slight hypesthesia develops in the median nerve distribution. Several investigators have noted that a significant percentage of patients initially present with decreased sensation involving the middle finger.[422,480] As the compression neuropathy advances, progressive loss of sensation, including impaired two-point discrimination, occurs.

A Tinel's sign may be elicited. It is produced by tapping gently over the volar wrist skin crease. A positive sign is manifested by a tingling or electrical shock sensation radiating into one or more of the median innervated digits. It most often is elicited in patients with severe CTS. However, it can produced in a number of patients without CTS. For this reason, it is not considered a sensitive or specific test for CTS. Nevertheless, when Tinel's sign is positive in the context of other characteristic signs and symptoms, it is supportive of the diagnosis. The Phalen's test is produced by placing the wrist in full flexion for 30 to 60 seconds. Symptoms will be exacerbated by this maneuver in a positive test. Phalen[480] found the test positive in 80% of 484 hands tested. Although a number of patients with definite CTS may not have a positive Phalen's test, when it is positive, it is a relatively reliable sign.

Dawson and colleagues[422] have grouped patients with CTS into three categories based on the severity of their symptoms. This grouping permits clearer identification of the patterns of presentation and thus aids in diagnosis. It also offers guidelines for treatment and prognostication.

Group I. Patients in Group I present with mild symptoms of intermittent numbness, tingling, and pain the median nerve distribution. They have nocturnal exacerbation of their symptoms and may be awakened from their sleep by pain. They may also relate that their symptoms are increased with driving, holding up a newspaper to read, and with manual labor, particularly sewing or other tedious hand activities. They have a positive Flick sign, which is manifest by vigorous shaking of their affected hand to "get the circulation going" with momentary, partial relief of their symptoms. Symptoms are initially sporadic, followed by increasing frequency. The physical examination is essentially normal, except perhaps for a positive Tinel's sign at the wrist or a positive Phalen's sign.

Group II. Group II CTS patients complain of persistent symptoms. Pain is their primary complaint. They also have hypesthesia, a sense of clumsiness along with actual decrease in dexterity with fine motor tasks. They have prominent nocturnal exacerbations and characteristically experience a notable increase in symptoms with manual activities. They complain of numbness, burning pain, and a sense of swelling in their hands. They usually have positive Flick, Tinel's, and Phalen's signs. They may rub their hands together or place the affected hand under water in an effort to obtain relief. On examination, these patients have objective weakness in thumb abduction and opposition, and may show thenar atrophy. They typically have sensory impairment in the median nerve distribution and possible vasotrophic changes.

Group III. Group III CTS patients have the most severe symptoms. They usually have longstanding complaints of painful dysesthesias and impaired dexterity with associated functional disability. Examination reveals severe sensory loss, including loss of two-point discrimination, and skin atrophy. They have marked thenar muscle wasting with significant weakness of thumb abduction and opposition. Prognosis is considered very poor in patients who present with these symptoms, regardless of therapeutic intervention.

Electrodiagnosis. Simpson[481] is credited as being the first investigator to demonstrate focal slowing of median nerve conduction across the wrist. In doing so, he provided the first neurophysiologic evidence of entrapment neuropathies. Nerve conduction across the wrist is a highly sensitive means of confirming the clinical diagnosis in most patients. In some cases, conduction studies across the wrist detect abnormalities in asymptomatic patients or in patients who have CTS but present with confusing symptoms, such as isolated shoulder or neck pain.

Median sensory nerve conduction across the wrist is a sensitive electrophysiologic means of diagnosing CTS.[422,482] The orthodromic stimulation technique involves use of ring electrodes, typically on the index or middle fingers, for stimulation. The active and reference surface electrodes are placed just proximal to the wrist crease with 14 cm conventionally used as the distance between stimulating and pickup electrodes in an adult. The antidromic technique involves stimulation proximal to the wrist crease with pickup of the sensory potential with ring electrodes on the index or middle fingers. The distance between the site of stimulation and the active electrode is again, conventionally, 14 cm. Although the latter technique characteristically results in larger sensory amplitudes, it also has the potential disadvantage of concomitant stimulation of a muscle action potential at the wrist, which may distort the sensory response.

More sensitive nerve conduction tests to detect CTS do exist. One particularly sensitive method is to compare the median vs ulnar sensory latencies to the ring finger in the same hand.[483]

The technique touted as being the most sensitive is comparison of the median and ulnar latencies with palmar stimulation. Palmar stimulation of the median and ulnar nerves generates a mixed nerve action potential for each nerve. These potentials are recorded at the wrist for comparison.[484] One other sensitive test is comparison of the median sensory latency across the wrist to the thumb with the radial sensory latency to the same digit.[485,486] Kimura[487] has performed serial stimulation from midpalm to distal forearm at 1-cm increments, known as the "inching" technique. Normally, median sensory axons show a predictable latency change of 0.16 to 0.20 ms/cm from one stimulation point to another. A localized latency increase across a 1-cm segment significantly greater than the other segments is suggestive of a focal median neuropathy.[160] Kimura[487] noted that the increase in latency in CTS occurred most often between 2 to 4 cm distal to the proximal border of the transverse carpal ligament. Identification of focal pathology rather than more diffuse involvement of the median nerve helps distinguish CTS from a distal neuropathy involving digital nerves, such as sometimes found in diabetics.

A reduction in amplitude of the SNAP may provide evidence to support the presence of CTS. Although not as sensitive as delayed conduction and of no localized value, reduced amplitude or absence of the SNAP does frequently occur in CTS. The range of normal amplitudes for the median SNAP is large, however, so it is advocated that the ratio of the median SNAP amplitude to that of the ulnar SNAP amplitude be obtained instead.[422]

The incidence of prolonged median motor distal latency is high in CTS, but it is not as sensitive as median sensory conduction across the wrist.[482,488] Segmental stimulation of median motor axons across the wrist can be done; however, this technique is technically more challenging than segmental stimulation of median sensory axons described above because the recurrent course of the thenar nerve anatomically varies among subjects.[489] A decrease in the amplitude of the median motor action potential does occur in CTS, but is not a sensitive indicator. Temporal dispersion of the action potential is also seen in some cases. Slowing of median motor nerve conduction in the forearm segment does not necessarily exclude CTS. It can be observed in more severe cases of CTS.[490]

The median NCS results on the clinically affected side may be compared with corresponding studies on the opposite side; however, because of the high incidence of bilateral involvement, this technique may have limited usefulness in improving the sensitivity of conduction studies in CTS.

Residual latency refers to the calculated time difference between the measured distal latency of a motor nerve and the expected distal latency. It is calculated by dividing the distance between the stimulus cathode and the active recording electrode by the maximum conduction velocity measured in a more proximal segment of the nerve. The residual latency is derived from the combination of the neuromuscular transmission time and the slowing of conduction in the terminal axons due to decreasing diameter and unmyelinated segments. The residual latency is increased in patients with CTS.[491]

Electromyography may be useful in identifying electrical evidence of motor axonal degeneration, but it is less helpful than the NCSs for diagnosing

CTS. More advanced cases of CTS may show increased insertional and spontaneous activity (fibrillations and positive sharp waves) in median innervated hand intrinsic muscles consistent with muscle membrane instability, along with a decreased recruitment pattern of motor unit action potentials, suggestive of denervation activity.

Current practice recommendations by the AAEM for electrodiagnosis of CTS suggest that sensory conductions of the median nerve across the wrist reflect a high degree of clinical certainty and should be standard practice. If the median sensory conduction is abnormal, one other sensory nerve in the symptomatic limb should be tested for comparison. If the median sensory conduction distance is greater than 8 cm and the latency results fall in the normal range, additional studies are necessary. A median conduction across the wrist of a distance less than 8 cm or comparison of the median conduction with radial or ulnar conduction in the same limb should be standard practice. Motor conduction studies of the median nerve, recording over the thenar eminence with comparison to one other motor nerve in the symptomatic limb is also recommended. It is felt however, that the motor conduction study reflects only moderate clinical certainty. An EMG of median innervated muscles of the thenar eminence is felt to be of unclear clinical utility and is left as an option at the electromyographer's discretion.[492]

Treatment. Conservative management of CTS is advocated for patients with mild or intermittent symptoms. Relative rest from the potentially precipitating or exacerbating activity, volar splinting of the wrist in neutral, and NSAIDs are often the initial measures used and are frequently helpful (Figure 9-65). Additional nonsurgical forms of treatment include local steroid injection of the wrist and a short course of oral steroids or a trial of diuretics, or both. Although pyridoxine at 100 to 200 mg per day has been prescribed, its actual benefits are inconclusive.[493,494] Doses as low as 300 mg/d have been shown to cause a progressive generalized sensory neuropathy.[495]

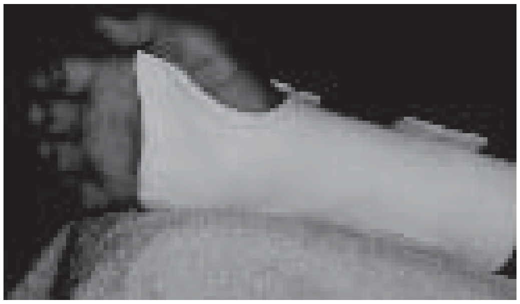


Fig. 9-65. Volar wrist hand orthosis (resting hand splint) may be very effective in the treatment of mild to moderate carpal tunnel syndrome.

Local steroid injections are used for patients with persistent but mild or intermittent symptoms, as well as for patients with painful symptoms who are considered poor surgical risks. Steroid injections are most successful in patients with minimal or intermittent symptoms and least successful in those with persistent numbness and thenar atrophy. Thus, injections are generally contraindicated in patients with significant or persistent sensory loss and thenar atrophy and weakness.[422] Green[496] found that a positive response to steroid injection is an excellent predictor of a successful surgical response. An acceptable method is to mix 1 to 2 mL of 1% lidocaine with 20 mg of methylprednisolone and inject this preparation proximal to the wrist crease and ulnar to the palmaris longus tendon. Care is taken to avoid direct injection into the median nerve or the transverse carpal ligament. Partial or complete relief usually occurs within 3 days. If no relief or exacerbation of symptoms occur, do not reinject. If partial relief occurs, consider a repeat injection. Do not inject more than four times due to the increased potential for tendon rupture and direct median nerve injury.[422]

When performed correctly, surgical management of CTS is generally highly successful. Absolute indications include failure of conservative management in a patient who is not a poor surgical risk or who manifests clinical evidence of thenar atrophy and weakness, or both. Persistent, longstanding sensory loss is considered a relative indication for surgical intervention.[422]

Postoperatively, a bulky hand dressing and wrist splint are applied. Most patients are permitted to begin to incompletely flex their digits within 48 hours. Finger flexion is gently progressed over the next several days. At 4 to 5 days, many patients are able to do light functional activities, but the wrist splint is maintained for up to 2 weeks, at which time the sutures may be removed. Strenuous activity is deferred for 2 to 3 months, but light clerical work can be progressively instituted after 2 weeks.

Acute CTS occurring in association with a fracture or fracture-dislocation, such as Colles' fracture, requires immediate closed reduction after documenting the median nerve deficit. If the symptoms of CTS persist or worsen after the reduction, immediate carpal tunnel release (CTR) is indicated. If symptoms of CTS occur after comminuted or unstable forearm or wrist fractures and persist greater than 24 to 48 hours despite operative management of the fractures, CTR is indicated. In the case of Volkmann's ischemic contracture secondary to a crush injury, CTR is done in combination with a more proximal fasciotomy.[422]

Ulnar Nerve Injuries

The ulnar nerve is derived from the medial cord of the brachial plexus, which formed from contributions from the C-8 and T-1 nerve roots. It carries both motor and sensory fibers. It supplies the flexor carpi ulnaris, the medial portion of the flexor digitorum profundus in the forearm, and the majority of the intrinsic muscles of the hand. The hand intrinsic muscles innervated include the following: first dorsal interosseous, adductor pollicis, deep head of the flexor pollicis brevis, flexor digiti minimi, abductor digiti minimi, opponens digiti minimi, volar interossei, dorsal interossei, and ulnar lumbricals (Figure 9-66). Sensory innervation includes the following: an articular branch to the elbow; the palmar cutaneous branch, which serves the palmar aspect of the hypothenar region; the dorsal cutaneous branch, which innervates the medial dorsum of the hand and the medial 1½ or 2½ digits; and the superficial branch, which serves the volar aspect of the medial 1½ digits.

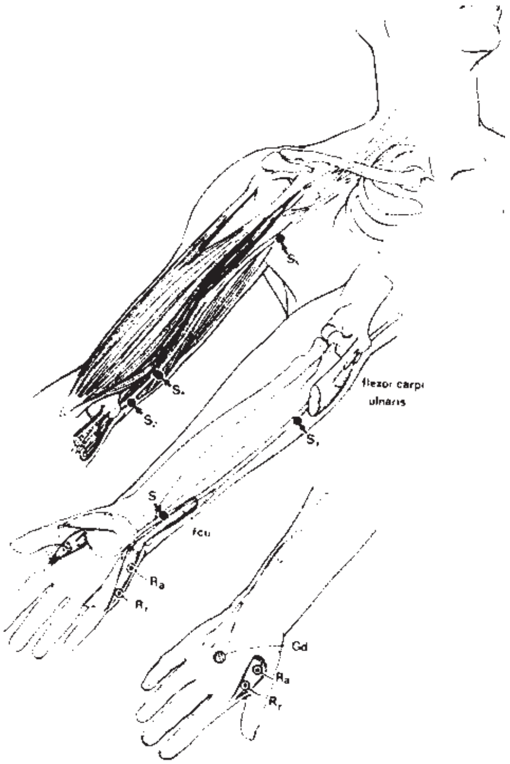


Fig. 9-66. The ulnar nerve with electrode placement for motor nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 152.

In the proximal upper arm, the ulnar nerve lies medial to the brachial artery. It remains in this relationship until the mid portion of the upper arm. At that point, it pierces the medial fascial septum and courses posterior to this septum in the posterior compartment. At the elbow, the ulnar nerve passes posterior to the medial epicondyle. It subsequently courses between the two heads of the flexor carpi ulnar, which constitutes the "cubital tunnel." It passes to the medial volar forearm area where it lies between the flexor carpi ulnaris and the flexor digitorum profundus. It lies medial to the ulnar artery in the distal forearm. In the distal forearm, the ulnar nerve is relatively superficial and courses between the flexor carpi ulnaris and flexor digitorum profundus tendons.

At the level of the wrist, the ulnar nerve passes through Guyon's canal, which is formed by connective tissue bounding the pisiform and the hook of the hamate. After passing through the canal, the ulnar divides into a superficial and a deep branch. The superficial branch supplies the palmaris brevis muscle and the skin of the hypothenar eminence and medial 1½ digits. The deep branch supplies the aforementioned intrinsic muscles of the hand.

Ulnar Nerve Injury at the Elbow

Ulnar nerve injuries and entrapment at the elbow are relatively common. Cubital tunnel syndrome and tardy ulnar nerve palsy are two terms used to describe the clinical presentation associated with ulnar nerve injury at the elbow. In order to clarify etiology, the term *cubital tunnel syndrome* is best restricted to those cases where the ulnar nerve is compressed by the aponeurosis of the flexor carpi ulnaris. *Tardy ulnar palsy*, on the other hand, is associated with antecedent trauma, precipitating joint deformity or recurrent nerve subluxation. Many clinicians, however, continue to use the term tardy ulnar palsy to describe any ulnar entrapment neuropathy at the elbow.

Anatomic considerations. The cubital tunnel at the elbow is formed by the following structures: its sides are formed by the two heads of the flexor carpi ulnaris, its floor by the medial ligaments of the elbow joint; the aponeurotic arch, which bridges the two heads of the flexor carpi ulnaris, forms the roof

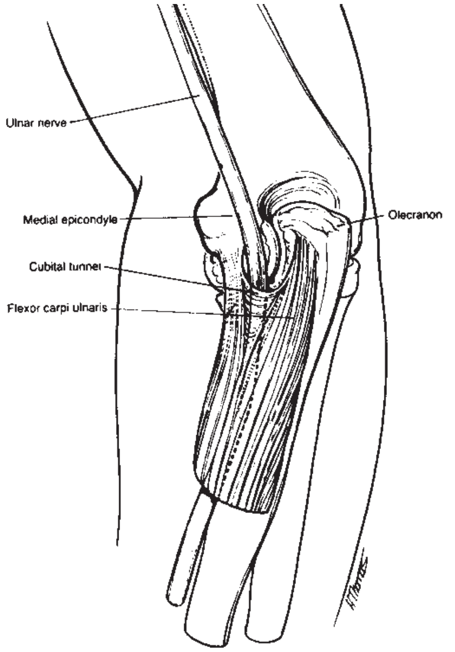


Fig. 9-67. The ulnar nerve passing through the cubital tunnel. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 30.

(Figure 9-67).[497] The cubital tunnel is a frequent site of entrapment.[498,499]

Clinical presentation. Patients with an ulnar nerve lesion at the elbow primarily present with complaints of intermittent hypesthesia in the ulnar distribution. Their symptoms are often exacerbated by sustained elbow flexion, particularly if also resting on the elbow, or by activities requiring repetitive flexion and extension of the elbow. Symptoms are generally intermittent at first. As symptoms progress, patients may experience nocturnal exacerbations with disturbance of sleep. They may awaken with elbow pain along with radiation of paresthesias from the elbow down the medial aspect of the hand to the little finger.

As noted above, sensory complaints are the initial symptoms. Impaired light touch and two-point discrimination, as opposed to pinprick and thermal sensations, are most often affected. The sensory deficit may involve the ulnar aspect of the palm, volar surface of the little finger and ulnar half of the ring finger, the medial dorsum of the hand, and the dorsum of the little and ring fingers. Note, however, that the ulnar nerve may supply the entire ring and ulnar half of the middle finger in about 20% of cases.[422] Most often, the sensory deficits and symptoms involve the superficial terminal branches to the palmar aspect of the little finger and medial half of the ring finger, along with the dorsum of these fingertips. In longstanding ulnar nerve entrapments, trophic changes may occur at the fingertips of the little or ring fingers. The medial cutaneous nerve branch of the brachial plexus serves the medial forearm up to a point proximal to the skin crease at the wrist. It is not involved in ulnar nerve lesions at the elbow.

Although most patients start with sensory complaints, an occasional patient will develop symptoms of weakness and impaired dexterity as their initial problem. In the majority of cases, progressive weakness of hand grip and pinch strength, as well as decreasing dexterity evolve slowly over time. With chronic compression, symptoms may be slowly progressive over months prior to developing objective motor deficits. With acute, traumatic compression injuries, sensory deficits and motor weakness may be immediate or variably delayed.

A positive Tinel's sign may be elicited at the elbow, typically with radiation down the medial aspect of the forearm. This, however, is a nonspecific sign that can be elicited in a number of patients without documented ulnar neuropathies. A more specific test for ulnar neuropathy is the elbow flexion test. In this test, elbow flexion reproducibly precipitates or exacerbates elbow pain or painful dysesthesias or both in the distribution of the ulnar nerve. The Tinel's sign and elbow flexion test are performed bilaterally for comparison.

Point tenderness, nerve subluxation, or nerve thickening may be palpable at the elbow. If the tenderness is detected several centimeters proximal to the medial epicondyle, it may be due to compression of the ulnar nerve by the arcade of Struthers or within the medial intermuscular septum. Tenderness elicited immediately posterior to the medial epicondyle is typically secondary to trauma. Tenderness reproduced 2 to 3 cm distal to the medial epicondyle in the region of the flexor carpi ulnaris aponeurosis is suggestive of cubital tunnel syndrome. If the tenderness is obtained even more distal, that is, greater than 3 cm distal to the medial epicondyle, suspect ulnar compression by the deep aponeurosis of the flexor carpi ulnaris.[422]

Subtle weakness may manifest as lateral instability of the index finger and increasing abduction and progressive weakness of the little finger. Weakness of ulnar innervated hand intrinsic muscles is also reflected by flexion contracture of the proximal IP joint of the little finger or ring finger, or both, along with hyperextension of the MCP joints of these digits.

Ulnar nerve lesions at the elbow may significantly affect a patient's fine motor function. The pinch strength between the thumb and fingers may be reduced due to weakness of the adductor pollicis, ulnar innervated portion of the flexor pollicis brevis, and first dorsal interosseous. The adductor pollicis and flexor pollicis brevis muscles normally stabilize the thumb MCP joint. A positive Froment's sign occurs when a patient uses the action of the flexor pollicis longus muscle (active flexion at the thumb IP joint), to compensate for weakness of the first dorsal interosseous or adductor pollicis muscle or both when attempting to perform a lateral pinch maneuver.

In the presence of ulnar neuropathy at the elbow, there is weakness of the flexor digitorum profundus muscles of the little finger and ring finger, as well as weakness of the ulnar innervated hand intrinsics. This results in both decreased hand grip power and impaired coordination in fine motor tasks, especially those requiring precision. Power grasp is approximately 100 to 140 lbs of pressure in an adult male and 50 to 60 lbs in an adult female. The dominant hand normally generates 15% to 20% more pressure than the nondominant limb.[422] A patient's power grasp strength may drop to as low as 20% to 25% of normal in the presence of a severe ulnar nerve lesion.[422]

One reason why some patients lack coordination when performing fine motor tasks is that weakness of the interosseous muscles compels them to initiate digit flexion at the MCP joints with the long finger flexors rather than the interossei. As the interosseous weakness progresses, a patient may develop variable degrees of a claw hand deformity involving the ring and little fingers.

Etiology. Ulnar nerve lesions at the elbow may occur as the result of sustained external pressure, direct trauma, chronic subluxation, or bony or scar impingement.

As previously mentioned, cubital tunnel syndrome refers to entrapment of the ulnar nerve by the aponeurosis of the flexor carpi ulnaris. This syndrome should be entertained when ulnar sensory or motor deficits or both develop without a readily identifiable cause. The cubital tunnel narrows and the medial collateral ligament bulges medially with elbow flexion. This accounts for why repetitive or sustained elbow flexion is implicated as a precipitating or exacerbating factor in some patients with symptoms of ulnar nerve compression.

Classic tardy ulnar nerve may occur when there is chronic stretch of the nerve secondary to a cubitus valgus deformity. This elbow malalignment often occurs after a capitular fracture with arrest of lateral humeral epiphyseal growth. A cubitus valgus deformity and tardy ulnar nerve palsy may also occur following a supracondylar fracture.[422] A compressive lesion at the elbow may affect different fascicles of the ulnar nerve. The most commonly affected are the fascicles carrying the fibers to the terminal digital nerves and intrinsic hand muscles rather than to the forearm muscles.[500] Fibers destined for the flexor carpi ulnaris are more lateral and posterior than the fibers to more distal muscles and are, therefore, more protected against trauma or compression.[422]

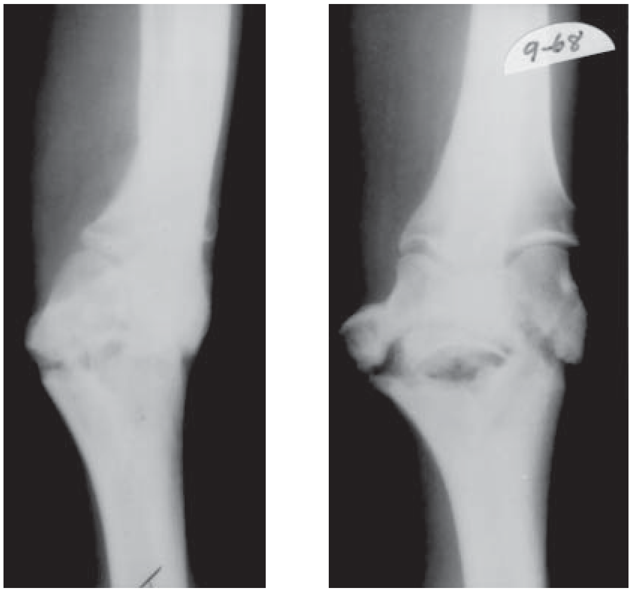


Fig. 9-68. Comminuted intracondylar distal humeral fracture, which may be associated with ulnar nerve injury. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

External pressure from trauma (Figure 9-68) or bony or soft tissue compression at the elbow, can be either acute or chronic and may be recurrent or sustained. A host of etiologies have been identified as a cause of ulnar neuropathy at the elbow[411,422,501–511]:

- Recurrent trauma
- Direct, blunt trauma, including elbow fracture and/or dislocation
- Recurrent subluxations
- Cubitus valgus deformity
- Ganglia
- Gouty tophus
- Accessory or aberrant anconeus muscle

- Synovial cyst
- Callus formation secondary to elbow fracture
- Posttraumatic calcification
- Scar formation post dislocation or other trauma
- Anomalous vessels, fibrous bands, or tumors at the elbow

Ulnar neuropathy at the elbow has been associated with rheumatoid arthritis.[512] It has also been documented in bedridden patients.[422] Chan et al[513] discovered that ulnar neuropathy tended to occur most often on the nondominant side in laborers, presumably because of their tendency to rest on their nondominant elbows for support while performing with the dominant arm.

Differential diagnosis of ulnar neuropathy at the elbow includes exclusion of an ulnar nerve lesion at the wrist. In the latter case, normal or only mildly slowed nerve conduction velocity would be present across the elbow or in the forearm segment, while slowing across the wrist would be present. Also, the dorsal cutaneous NCS would be normal with wrist lesions in contrast to the abnormal ulnar sensory nerve conduction across the wrist. In more severe ulnar injury at the wrist, EMG abnormalities would be present in ulnar innervated hand intrinsics while sparing the ulnar innervated forearm muscles. TOS, C-8/T-1 radiculopathy, high (more proximal) ulnar nerve lesion, medial cord lesion (eg, secondary to a superior sulcus tumor), syringomyelia, and early amyotrophic lateral sclerosis may also require exclusion.

Electrodiagnosis. Simpson[481] has been credited with being the first to demonstrate that slowed conduction across the elbow segment is helpful in localizing an ulnar nerve lesion at the elbow. Slowing of the ulnar motor nerve conduction velocity across the elbow segment is the most specific diagnostic criterion for identifying an ulnar compression neuropathy at the elbow.[498,503,514,515] Although it may be helpful to compare the conduction velocity across the elbow segment with that obtained in the above- and below-elbow segments, the absolute conduction velocity is felt to be a more sensitive indicator of ulnar conduction abnormality than the relative velocity.[503,515] The degree of slowing, however, does not necessarily correlate with severity.[422]

Focal demyelination injury of the ulnar nerve at the elbow will cause temporal dispersion of the CMAP when stimulation occurs proximal to the lesion. A decrease in amplitude may also be seen. A decrease in amplitude of greater than 10% is felt to be abnormal.[515] In a study by Pickett and Coleman,[516] a decrease in amplitude of greater than 25%, with stimulation proximal to the elbow compared to distal to the elbow, was the best criterion for confirming an ulnar nerve conduction deficit across the elbow.

Whenever assessing amplitude changes with median and ulnar NCSs, it is important not to overlook the possible presence of a Martin-Gruber anastomosis, seen in up to 20% of the population (Figure 9-69). In this anomaly, some nerve fibers from the median nerve cross over to the ulnar nerve in the forearm and course with the ulnar nerve fibers through Guyon's canal. This process will result in a larger ulnar CMAP amplitude with stimulation at the wrist rather than at the elbow, which, in turn, may cause an erroneous assumption that there has been an amplitude drop due to a partial conduction block at the elbow. Assuming that there is not

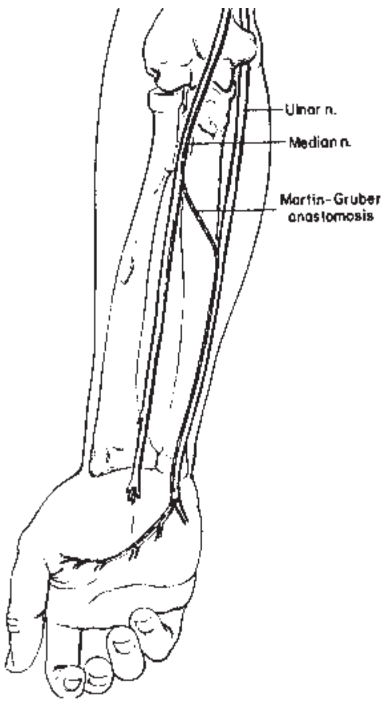


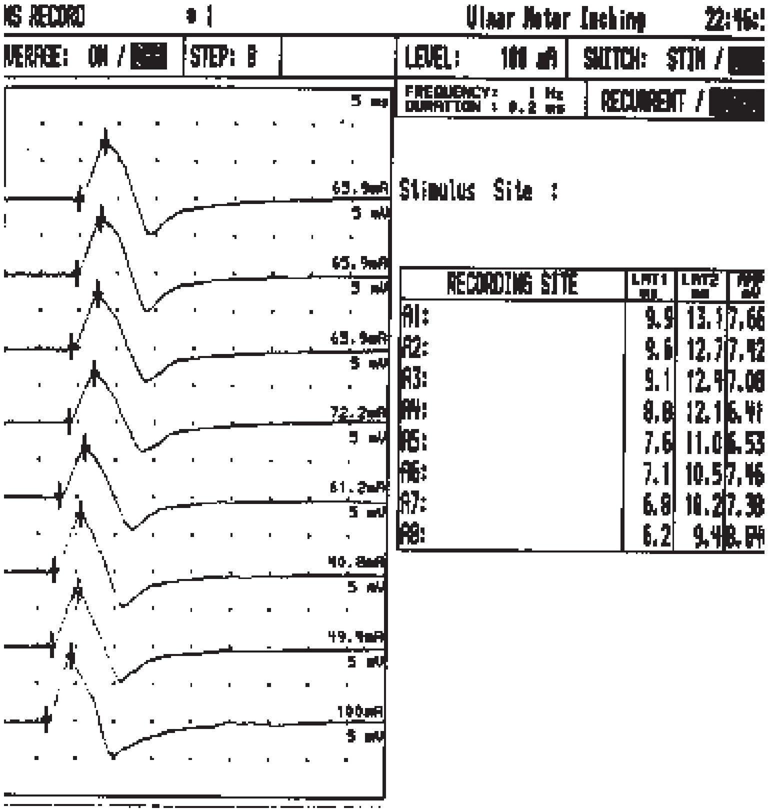
Fig. 9-69. Martin-Gruber anastomosis. Fascicles from the median nerve cross over in the forearm and run with the ulnar nerve. Stimulation of the median nerve at the wrist will generate a compound motor unit action potential (CMUAP) of lower amplitude than stimulation at the elbow. Stimulation of the ulnar nerve at the wrist will evoke a larger amplitude than stimulation around the elbow. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 27.

a concomitant ulnar neuropathy actually present at the elbow, a similar small CMAP amplitude would be obtained with stimulation below and above the elbow in the presence of a Martin-Gruber anastomosis.

When performing ulnar NCSs across the elbow, it is important to maintain a consistent elbow position as the length of the nerve changes with movement of the arm into different positions. While different angular elbow positions have been advocated, Bielawski and Hallet[517] observed no major difference in the ability to diagnose a lesion at the elbow between flexed or extended positions. They did, however, stress that the same technique should be used consistently by physicians in the same electrodiagnostic laboratory. It is also crucial to avoid too short an interstimulus distance in order to prevent inaccurate conduction velocity calculations. Some argue that the cathode for below-elbow stimulation should be at least 4 cm distal to the medial epicondyle and the above-elbow stimulus site at least 10 cm proximal to the below-elbow site.[422] Adherence to this technique helps to ensure that the focal site of compression is included in the across-the-elbow segment and that a sufficiently long segment is assessed. It is particularly important if cubital tunnel syndrome is suspected, as the site of nerve constriction is felt to be 1.5 to 3.5 cm distal to the medial epicondyle.[499] Surface pickup over the abductor digiti minimi muscle is the most common technique for ulnar motor studies. However, it may be preferable to place the surface pickup electrode over the first dorsal interosseous muscle instead, because motor fibers to this muscle are more likely to be involved in an ulnar nerve lesion at the elbow, and thus more likely to demonstrate conduction slowing or block.[500]

A decrease in the ulnar SNAP amplitude obtained distally across the wrist confirms the presence of an ulnar nerve lesion and correlates with severity, but, as an isolated finding, is of no localizing value.[498,503] If the ulnar dorsal cutaneous SNAP is unobtainable or its amplitude reduced, particularly if its latency is within normal limits, it is supportive of a more proximal ulnar nerve lesion. However, it cannot be used to definitely localize the site to the elbow area. Because the amplitude of a SNAP reflects the number of functioning nerve fibers, a decreased SNAP amplitude indicates more severe injury and possibly a poorer prognosis.[422,500]

Recently, a short segment incremental study of the ulnar nerve across the elbow has been shown to be a sensitive tool in further localizing areas of entrapment[518] (Figure 9-70). Stimulation at 1-cm in-



MS RECORD #1 Ulnar Motor Inching 22:46:

AVERAGE: ON / STEP: B

LEVEL: 100 mA SWITCH: STIM /

FREQUENCY: 1 Hz
DURATION : 0.2 ms

RECURRENT /

Stimulus Site :

RECORDING SITE	LAT1 ms	LAT2 ms	[illegible]
A1:	9.9	13.1	7.66
A2:	9.6	12.7	7.42
A3:	9.1	12.9	7.08
A4:	8.8	12.1	[illegible]
A5:	7.6	11.0	6.53
A6:	7.1	10.5	7.46
A7:	6.8	10.2	7.38
A8:	6.2	9.4	[illegible]

Fig 9-70. Short segment incremental study of the ulnar nerve across the elbow. A 1.2-ms latency delay was observed 2 cm distal to the medial epicondyle. The finding suggested compression at the cubital tunnel. Tracing courtesy of CPT Ronald T. Stephens, M.D., Physical Medicine and Rehabilitation Electrodiagnostic Laboratory, Walter Reed Army Medical Center, Washington, DC.

crements along the path of the ulnar nerve, extending above and below the elbow, enabled the differentiation of entrapment at the retroepicondylar groove from entrapment at the humeroulnar aponeurotic arcade (cubital tunnel). A latency change over a 1-cm segment of greater than 0.4 ms was found to be significant. Latency delays at the medial epicondyle reflected entrapment at the retroepicondylar groove. Slowing 1 to 3 cm distal to the medial epicondyle was consistent with compression in the cubital tunnel. Latency prolongation at a focus more distal than 3 cm was felt to be secondary to entrapment at the exit from beneath the flexor carpi ulnaris.[518]

Electromyography is not as sensitive as NCSs in identifying and localizing an ulnar nerve lesion at the elbow. However, it can be a useful adjunct to these studies, particularly if EMG abnormalities suggestive of denervation activity are found in both the ulnar innervated forearm and hand muscles. Electromyography may also be helpful in excluding a C-8/T-1 radiculopathy.

Treatment. Conservative management is appropriate for patients with mild symptoms. This includes not resting on the elbow (especially when flexed), as well as avoidance of sustained or extreme elbow flexion and activities requiring repetitive flexion and extension of the elbow. Some advocate use of an elbow splint, such as orthoplast splints or long-arm, bivalved casts to maintain the elbow in extension or partial flexion, but tolerance and compliance may be a problem. A well-cushioned elbow pad is well tolerated and minimizes the opportunity for inadvertent trauma to the elbow. A lumbrical bar orthotic is prescribed for selected patients showing signs of developing a claw-hand deformity or who already demonstrate this deformity but have not yet developed fixed contractures. This particular orthotic is helpful in preventing progression of the deformity, particularly if used in combination with daily range-of-motion exercises of the involved digits.

Various surgical procedures are available to treat ulnar neuropathy at the elbow. These include release of the flexor carpi ulnaris aponeurosis, medial epicondylectomy, and anterior transplantation of the nerve to a subcutaneous position or under the flexor and pronator muscles.[422] Surgical management is indicated for patients with persistent or progressive signs and symptoms of ulnar neuropathy, particularly progressive weakness, despite a trial of conservative management.

In the event cubital tunnel syndrome is identified, a cubital tunnel release may be sufficient to relieve symptoms. Cubital tunnel release involves the release of the flexor carpi ulnaris aponeurosis. When indicated, it has the advantage of being a relatively simple procedure with low morbidity. It can be done under local anesthesia and does not require postoperative splinting. It is not, however, recommended for patients with osseous or joint pathology at the elbow or for those with congenital subluxation of the nerve.[422]

For patients who fail to receive satisfactory results from a simple release, a medial epicondylectomy or anterior transplantation may be warranted.[519] Excision of the medial epicondyle removes a potential or actual cause of nerve compression, requiring less dissection with less chance of injuring nerve branches than the anterior transplantation procedure. The disadvantage is that the ulnar nerve is still in a subcutaneous position and, therefore, remains vulnerable to trauma.

Postoperatively, the elbow is protected with a posterior splint for two weeks, at which time gently progressive range-of-motion exercises are instituted. Results have generally been good with this procedure.[422,520–522]

Anterior transplantation is designed to remove the ulnar nerve from compressive forces in the cubital tunnel. The nerve is placed in a less vulnerable, anterior position. This position also decreases the tension on the nerve during elbow flexion and places the nerve in a well-vascularized intermuscular bed. Anterior transplantation is, however, a more challenging procedure than either the simple release or medial epicondylectomy. Since it involves use of a large skin flap, there is a risk of injury to the posterior branch of the medial antebrachial cutaneous nerve.

Except in those rare cases where isolated cubital tunnel syndrome is identified and simple cubital tunnel release is sufficient, Dawson and colleagues[422] prefer medial epicondylectomy to either the subcutaneous or submuscular anterior transposition procedure. It is specifically preferred because of its relatively good success rate and because it requires less dissection and devascularization of the nerve.

Ulnar Neuropathy at the Wrist

Anatomic considerations. At the level of the wrist, the ulnar nerve passes through a closed space referred to as Guyon's canal (Figure 9-71). Within the canal, the nerve passes over the transverse carpal ligament. The volar carpal ligament roofs the nerve at this point. The pisiform and the hook of

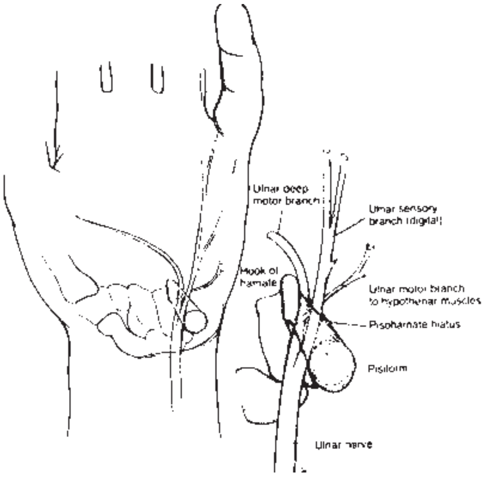


Fig. 9-71. Guyon's canal and its relationship to the distal branches of the ulnar nerve. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 31.

the hamate form its bony margins. The ulnar artery and vein also pass through Guyon's canal. The ulnar nerve branches into its terminal superficial and deep branches within the canal.

The superficial terminal branch of the ulnar nerve supplies the palmaris brevis, innervates the skin on the distal medial aspect of the palm, and gives off two palmar digital nerves. In the majority of cases, the ulnar nerve provides sensory innervation to the little finger and the medial half of the ring finger. In a minority, sensation is provided to the little and ring fingers and the medial half of the middle finger. The deep terminal branch supplies the hypothenar muscles (abductor digiti minimi, flexor digiti minimi brevis, and opponens digiti minimi) and then dives into the palm under the flexor tendons to supply the palmar and dorsal interossei, the third and fourth lumbricals, a portion of the flexor pollicis brevis, and finally the adductor pollicis.

The palmar cutaneous branch of the ulnar nerve arises at 7 cm proximal to the wrist, descends near the ulnar artery, pierces the deep fascia, and supplies the skin over the hypothenar eminence. The dorsal cutaneous ulnar nerve branch arises 5 to 10 cm above the wrist, passes posteriorly deep to the tendon of the flexor carpi ulnaris, pierces the deep fascia, and supplies the skin on the medial dorsum of the hand, as well as the dorsum of the little and ring fingers and the medial dorsum of the middle finger (Figure 9-72).

The most common site of ulnar nerve compression at the wrist is within Guyon's canal.[422,523] The nerve is also vulnerable to compression just proximal or distal to the canal. Rarely, an isolated compression injury of the dorsal ulnar cutaneous nerve may occur proximal to the wrist.

Clinical presentation. The distribution of sensory impairment and weakness is dependent on the level of ulnar nerve compression injury. If there is total ulnar sensory loss involving both the dorsal and palmar surfaces of the medial aspect of the hand, as well as the fourth and fifth digits, along with weakness of all ulnar innervated hand intrinsics, then a lesion proximal to the wrist is suspected.

A lesion within Guyon's canal will spare the sensory distribution of the palmar cutaneous branch and the dorsal ulnar cutaneous branch. If a lesion occurs proximal in the canal, however, it may cause sensory impairment in the distal medial aspect of the palm and the volar surfaces and tips of the fourth and fifth digits. It may also cause weakness of the hypothenar muscles, as well as the interossei, third and fourth lumbricals, flexor pollicis brevis (deep head), and adductor pollicis.

Compression injury isolated to sensory fibers within Guyon's canal can occur. In this case, sensation is preserved in the distribution of the dorsal ulnar cutaneous and palmar cutaneous branches, but sensory loss typically occurs on the volar aspect of the little finger and medial half of the ring finger. With a lesion more distal in the canal, at or distal to the hook of the hamate, innervation to

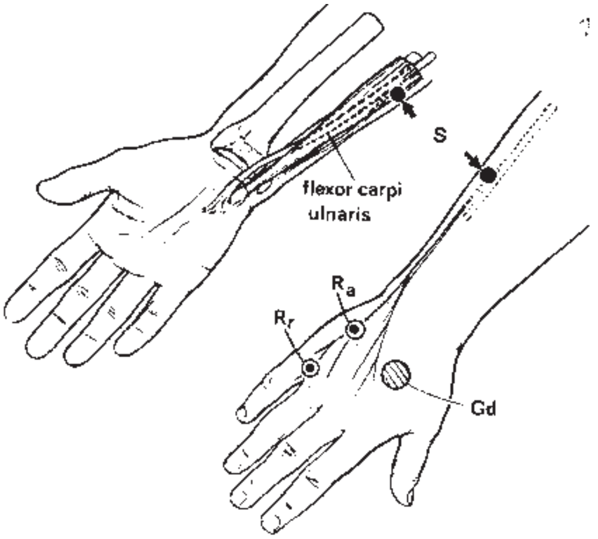


Fig. 9-72. The dorsal cutaneous branch of the ulnar nerve with electrode placement for sensory nerve conduction studies. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 178.

hypothenar muscles is preserved, but other ulnar-innervated hand intrinsic muscles will be weak.

A lesion of the deep palmar branch of the ulnar nerve, distal to Guyon's canal, will spare the hypothenar muscles and all ulnar sensory fibers. However, it will cause weakness of the remaining ulnar-innervated hand intrinsics, including the fourth and fifth lumbricals, interossei, flexor pollicis brevis (deep head), and adductor pollicis muscles.

Based on the aforementioned potential sites of ulnar nerve compression at or near the wrist, patients may have mixed motor and sensory deficits, isolated sensory loss, or pure motor deficits. Wrist pain with radiation proximal or distal may occur. There may be nocturnal exacerbations and cold intolerance. Clinical examination may reveal local swelling, induration, and a palpable mass, or a positive Tinel's sign or both.

Etiology. A multitude of causes of ulnar nerve compression at or distal to the wrist have been reported. Extrinsic pressure in the form of repetitive trauma, such as experienced in some occupations or sports activities, or chronic compression secondary to masses, have been documented to cause ulnar neuropathies of the wrist and hand.

Hunt,[524] in 1908, was the first to identify occupational trauma as the cause of an ulnar compression neuropathy, specifically a neuritis of the deep palmar branch of the ulnar nerve. Chronic compression neuropathy of the ulnar nerve may occur in pipe cutters, metal polishers, mechanics, and professional cyclists.[422] In the cyclists, ulnar neuropathy has been attributable to chronic pressure from leaning on the handlebars.[525,526] A variety of causes of ulnar nerve compression at or distal to the wrist have been reported[527–542]:

- Occupational trauma
- Bicycling (handlebar palsy)
- Edema
- Wrist fracture
- Ganglion
- Calcium deposits
- Tumor
- Fibrous band
- True aneurysms
- False aneurysms
- Rheumatoid synovial cyst

An alternate list of causes has been documented by Shea and McClain[543] in order of highest to lowest occurrence among 137 patients:

- Ganglia
- Occupational Neuritis
- Laceration
- Ulnar artery disease
- Arteritis
- Thromboangiitis
- Fracture of carpal bones
- Soft tissue contractures
- Fracture of metacarpal bones
- Fracture of radius
- Aberrant muscles
- Neurolemoma
- Anomalous relationship of nerve to Guyon's canal
- Pisiform bursitis
- Carpal osteoarthritis
- Accessory ossicle
- Dislocation of distal ulna
- Lipoma
- Hemophiliac cyst
- Dislocation of pisiform

Differential diagnosis of ulnar neuropathy at the wrist includes C-8, T-1 radiculopathy, TOS, amyotrophic lateral sclerosis, ulnar neuropathy at the elbow or in the forearm, CTS, and isolated fractures of the carpal bones. Ulnar neuropathy in the wrist area may be due to fractures of the metacarpals, pisiform, or body or hook of the hamate. Thus, these fractures should be excluded when a patient has a history of trauma to the wrist, point tenderness over the affected carpal or metacarpal bones, and ulnar nerve sensory and especially motor deficits. In the event of a fracture of the pisiform or hook of the hamate, special radiologic views, including a carpal tunnel view, lateral tomograms, or oblique views of the hand, will be necessary to identify these fractures as, conventional radiograph views are typically negative.

Electrodiagnosis. The most common ulnar motor NCS performed across the wrist is stimulation at the wrist with surface pickup at the abductor digiti minimi. However, the most common site of ulnar nerve compression in the wrist area involves the deep palmar branch distal to the branch to the abductor digiti minimi. Thus, nerve conduction to the abductor digiti minimi alone is not sufficient to exclude an ulnar motor neuropathy in the wrist region. Motor nerve conduction across the wrist to the first dorsal interosseous, however, would demonstrate a prolonged latency or unobtainable evoked response in the presence of compression of the deep palmar branch. Electromyography, in this case, would be normal in the hypothenar muscles but show evidence of denervation activity in other ulnar-innervated hand intrinsics.

In more proximal lesions within Guyon's canal, ulnar motor nerve conduction to both the abductor digiti minimi and the first dorsal interosseous would be absent or prolonged and EMG evidence of denervation activity could be detected in all ulnar-innervated hand intrinsics.

If an ulnar nerve lesion is sufficiently proximal within Guyon's canal or proximal to Guyon's canal, then both the deep and superficial terminal branches of the ulnar nerve may be involved. In this case, both ulnar motor and sensory NCSs would be abnormal. Ulnar sensory studies, either orthodromic or antidromic, would reveal a prolonged latency or absent SNAP. If obtainable, the SNAP may also be reduced in amplitude. Ulnar nerve lesions at or distal to the wrist spare the dorsal ulnar cutaneous nerve. Thus, sensory nerve conduction of this nerve may have localizing value.

Isolated compression neuropathy of the ulnar sensory fibers is the least common type of nerve lesion at the wrist.[543] If present, only the ulnar sensory nerve conduction across the wrist is abnormal, manifest by a prolonged latency, reduced SNAP amplitude, or unobtainable response.

The dorsal ulnar cutaneous branch neuropathy is uncommon. When it occurs, it is usually secondary to blunt trauma or lacerations. It is less likely to be injured than the superficial radial nerve since it lies in a more protected position and because it is subject to less iatrogenically induced surgical trauma. In the event this nerve is injured, isolated abnormality of the dorsal ulnar cutaneous NCS will be present, manifest by a prolonged latency, reduced SNAP amplitude, or unobtainable response.

Treatment. Treatment of ulnar compression injuries in the region of the wrist depend on the site, etiology, and duration of the lesion. Conservative management is reserved for mildly symptomatic patients. This includes relative rest in the event of isolated trauma or avoidance of repetitive or chronic compressive trauma. In certain predisposing occupations, relative rest or modifications, such as padded gloves when performing work, may be sufficient to relieve symptoms.

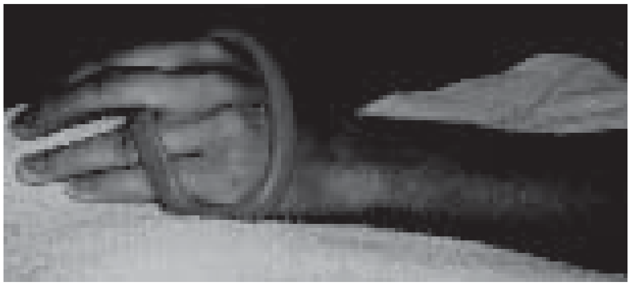


Fig. 9-73. Lumbrical bar hand orthosis compensates for weakness of the ulnar innervated lumbrical muscles and limits metacarpophalangeal hyperextension, placing the fingers in a functional position.

In the event a patient experiences extrinsic trauma that does not respond to conservative management, surgical intervention may be indicated. This may include exploration and decompression or neurolysis, or both. Tumors and ganglia are removed. A fracture of the hook of the hamate is excised in combination with decompression and neurolysis of the nerve. A fracture of the pisiform may also require excision.

If clawing of the lateral two digits occurs secondary to weakness of ulnar-innervated intrinsic muscles, a lumbrical bar orthotic may be helpful in preventing fixed contractures (Figure 9-73). This is most successfully employed when used in conjunction with daily range-of-motion exercises of the affected digits.

NERVE INJURIES AND ENTRAPMENT NEUROPATHIES IN THE LOWER EXTREMITY

Lumbosacral Plexopathy

Anatomic Considerations

The lumbar plexus is produced by the union of the ventral rami of the first three lumbar nerves and the greater part of the fourth. There is a contribution from the subcostal nerve as well. The lumbar plexus lies anterior to the lumbar vertebral transverse processes. It is embedded in the posterior aspect of the psoas major muscle. The lower part of the ventral ramus of the fourth lumbar nerve joins the ventral ramus of the fifth to form the lumbosacral trunk.[473]

The sacral plexus is formed by the lumbosacral trunk and the ventral rami of the first three sacral nerves and the upper part of the fourth sacral ramus. The sacral plexus gives rise to multiple branches prior to the greater sciatic foramen. Its remaining fibers emerge from the greater sciatic foramen as the sciatic nerve (Figure 9-74).[473]

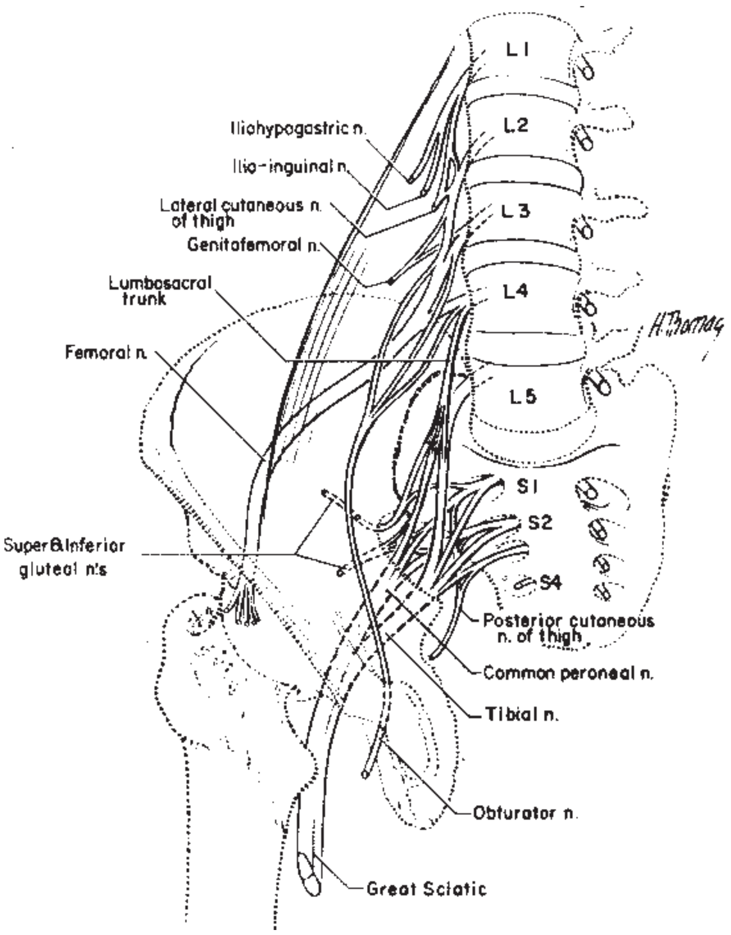

L1
L2
L3
L4
L5
S1
S2
S4
Iliohypogastric n.
Ilio-inguinal n.
Lateral cutaneous n. of thigh
Genitofemoral n.
Lumbosacral trunk
Femoral n.
Super & Inferior gluteal n's
Posterior cutaneous n. of thigh
Common peroneal n.
Tibial n.
Obturator n.
Great Sciatic

Fig. 9-74. The lumbar and sacral plexuses. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 47.

Clinical Presentation

Lumbosacral plexopathies usually occur secondary to trauma. Thus, onset of weakness is typically acute with variable distribution and severity of weakness and sensory deficits depending on the specific portion of the plexus injured. Hyporeflexia or areflexia may also be present in the affected limb at the patella or ankle or both, depending upon site and extent of nerve pathology. Bowel or bladder dysfunction may occur if fibers from the lower sacral nerve roots are injured.

Etiology

Lumbosacral plexopathies are uncommon. Two of the most common causes include pelvic fractures and retroperitoneal hemorrhage. Pelvic fractures usually injure the sciatic nerve with the peroneal component most significantly affected. However, fracture of the pelvic ring or a fracture near the sacroiliac joint may directly damage the plexus or one or more of its branches (Figure 9-75).[411,544,545] Such fractures have been recorded to occur as a complication of cancer or postradiation therapy.[546]

Electrodiagnosis

Electromyography is the most helpful electrodiagnostic test. Findings consistent with denervation activity can be demonstrated by sampling clinically weak muscles of the affected extremity. An EMG of the lumbosacral paraspinals, on the other hand, will be normal unless both nerve roots and plexus are concomitantly involved. If sensory deficits are present in the distribution of the lateral femoral

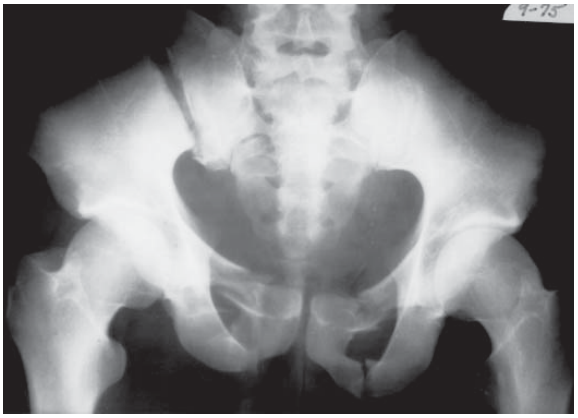


Fig. 9-75. Fractures of the left ileum and right and left ischial arches, associated with traumatic lumbosacral plexopathy. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

cutaneous, saphenous, or sural nerves, sensory NCS of these nerves may be supportive of a plexopathy if they are abnormal, assuming the clinical examination and EMG also suggest a plexopathy. The sensory action potentials of these nerves may be attenuated in amplitude or unobtainable in the presence of a plexopathy affecting their nerve fibers. The soleus H-reflex may also be supportive if prolonged or unobtainable, assuming an S-1 radiculopathy has been excluded clinically or electrodiagnostically.

Sciatic Neuropathy

Anatomic Considerations

The fourth lumbar to the third sacral roots contribute to the formation of the sciatic nerve (Figure 9-76). The nerve lies anterior to the piriformis in the lesser pelvis. In the majority of cases, the nerve passes below the piriformis muscle. In a small percentage, it passes through the piriformis. The nerve then courses laterally beneath the gluteus maximus and passes along the posterior surface of the ischium. It then runs midway between the ischial tuberosity and the greater trochanter and downward over the gemelli, the obturator internus tendon, and the quadratus femoris. The latter muscle separates the sciatic nerve from the hip joint. The nerve then enters the posterior thigh beneath the lower border of the gluteus maximus.

The sciatic nerve courses down the middle of the posterior thigh. At a point just above the apex of the popliteal fossa, it is overlapped by the margins of the biceps femoris and semimembranosus muscles. In the great majority of cases, the sciatic nerve divides into its tibial and peroneal branches near the apex of the popliteal fossa. In a smaller percentage, the nerve divides into these two branches more proximally. In rare cases, the tibial and peroneal branches arise independently from the sacral plexus and course in parallel until they reach the apex of the popliteal fossa where they divide.

The sciatic nerve supplies an articular branch to the hip. It also supplies branches to the semimembranosus, semitendinosus, ischial head of the adductor magnus, and to both heads of the biceps femoris. The branch to the short head of the biceps femoris actually arises from the peroneal portion of the sciatic nerve. All other posterior thigh muscles innervated by the sciatic nerve arise from the tibial division.

In injuries involving the sciatic nerve, the peroneal nerve fibers are more often and more severely

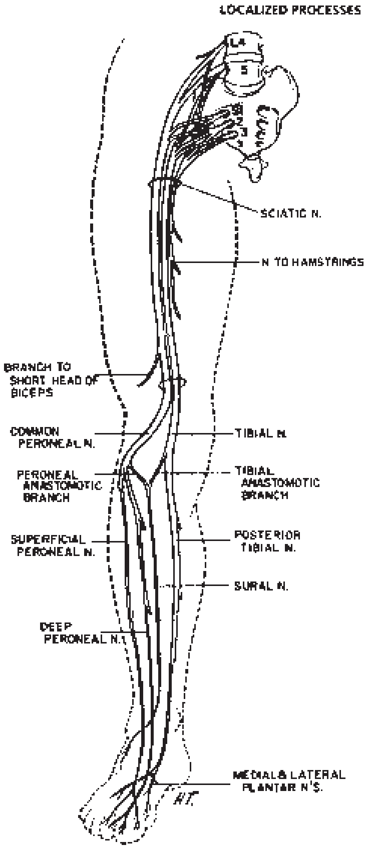


Fig. 9-76. The sciatic nerve with its distal branches. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 49.

affected than the tibial fibers. This is explained by the fact that the peroneal division contains fewer and larger funiculi and has less adipose tissue available to protect the nerve from trauma.[547]

Etiology

Entrapment of the sciatic nerve as it passes through the piriformis or between the piriformis and underlying gemelli, is a well known but clinically rare entity.[548] The piriformis syndrome is presumed to result from compression of the sciatic nerve between the two tendinous heads of the pirformis muscle. Irritation of the nerve may also occur with provocative positioning, especially flexion-adduction-internal rotation.

Other potential causes of sciatic neuropathies include retroperitoneal hemorrhage,[549] fibrous bands spanning the biceps femoris and adductor magnus,[550] and gluteal compartment syndrome.[551] Association with proximal femoral fracture and dislocation has been documented[552] and was observed during the Persian Gulf War (Figure 9-77). Association of sciatic neuropathies with acetabular fractures has been noted to be as high as 13%.[553] Recently, Kaplan and Challenor[554] observed the development of a sciatic neuropathy caused by the development of a posttraumatic osseous tunnel surrounding the nerve. Sciatic nerve palsies occur in association with total hip replacement in less than 2% of patients.[555] Its presence, however, significantly impacts on the rehabilitation and ultimate functional outcome of these patients. In Schmalzried's study, of 36 patients who sustained sciatic nerve injuries (followed for 24 months postsurgery), 64% had persistent mild neurologic deficits and 16% suffered from severe nerve injury.[556]

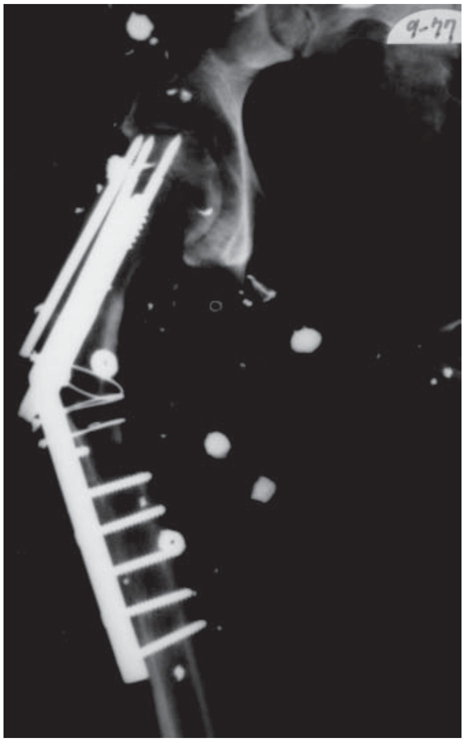


Fig. 9-77. Comminuted proximal femur fracture following open reduction internal fixation. The patient sustained a concomitant complete sciatic nerve injury.

Clinical Presentation

Proximal sciatic neuropathies may manifest in a number of ways. Pain in the gluteal region, radiating down the posterior aspect of the leg and calf are characteristic. In the piriformis syndrome, symptoms are aggravated by prolonged sitting and with resisted external rotation of the hip. Severe injury to the sciatic nerve will compromise control of the foot and ankle, severely affect knee flexion and hip extension, and variably ablate sensation below the knee, except in the medial malleolar region, which is supplied by the saphenous nerve.

Electrodiagnosis

Stewart's[557] criteria for piriformis syndrome can be generalized to other proximal sciatic neuropathies. Evidence of denervation in muscle supplied by the sciatic nerve including the hamstrings, tibial, and peroneal-innervated muscles may be observed. Muscles in the same myotomal distribution but innervated by the superior gluteal or inferior gluteal nerves are normal. Lumbar paraspinal muscles also show no evidence of denervation. The electromyographic studies are most helpful distinguishing the neuropathy from the much more common L-5 and S-1 radiculopathies. Nerve conduction studies are of lesser utility. Two techniques deserve mention. MacLean[558] developed a method for measuring latencies across the sacral plexus as well as the proximal sciatic nerve. The L-5 and S-1 roots are stimulated just medial and slightly caudal to the posterior superior iliac spine. Recording electrodes are placed on the abductor hallicus. Stimulation at the root level is followed by near nerve stimulation at the sciatic notch. Subtraction of the distal latency from the proximal latency establishes the latency across the proximal nerve segment. Normal latency obtained by MacLean[558] for the sciatic plexus/sciatic nerve was less than 5.3 ms (mean plus two standard deviations). Side-to-side latency differences should not be greater than 0.9 ms. Fishman and Zybert[559] have developed a provocative test that measures latency shifts in H-reflexes before and during flexion-adduction-internal rotation positioning of the hip.

Femoral Nerve Injury

Anatomic Consideration

The posterior divisions of the second, third, and fourth lumbar roots entwine at the level of the psoas muscle. Piercing the muscle, the femoral nerve courses between the iliacus muscle and the psoas tendon. The nerve then crosses the pelvic brim, beneath the inguinal ligament (Figure 9-78). Before entering the femoral triangle, motor branches to the quadriceps, sartorius, and pectineus separate off. Cutaneous sensory branches arborize across the anterior thigh. The saphenous nerve, the terminal branch of the femoral nerve, dives deep with the femoral vessels and penetrates the adductor canal. The nerve becomes superficial in the popliteal region and extends along the medial aspect of the leg, providing sensation down to the medial malleolus and occasionally to as distal as the great toe (Figure 9-79).

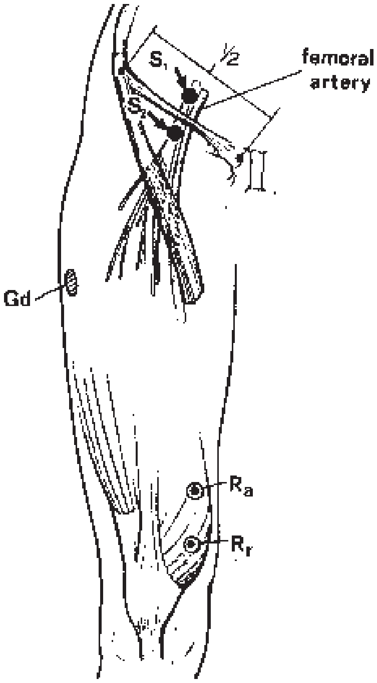


Fig. 9-78. The femoral nerve, passing beneath the inguinal ligament. Electrode placement for motor nerve conduction studies are noted. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 190.

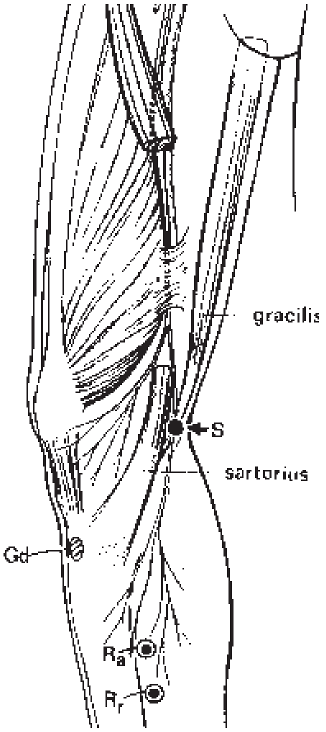


Fig. 9-79. The saphenous nerve, the terminal sensory branch of the femoral nerve, passing through the adductor canal, transmitting sensation from the medial aspect of the leg. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 193.

Etiology

Retroperitoneal hemorrhage, involving the iliopsoas muscle is a common cause of this uncommon injury (Figure 9-80). Associations with hemophilia and anticoagulation are obvious.[560,561] Traumatic injury to the iliopsoas with subsequent femoral nerve injury has been observed following efforts to regain balance during severe hyperextension at the hip.[562,563] Other related traumas have included falls, gunshot wounds, and pelvic fractures.[564] Iatrogenic femoral neuropathies have occurred following pelvic surgery, attributed to the surgery itself as well as to prolonged lithotomy positioning of the pa-

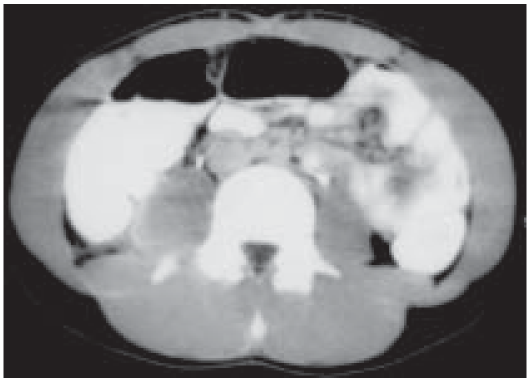


Fig. 9-80. Computed tomography of the pelvis revealing prominent asymmetry of the right psoas vs the left, consistent with psoas hemorrhage. Soft tissue density extending along the right ileum is the residua of retroperitoneal hemorrhage. Femoral nerve entrapment was associated with this traumatic injury.

tient.[564,565] Cases have also been reported of injuries occurring after total hip arthroplasty and femoral vessel catheterization.[566–568]

Clinical Presentation

Weakness of the knee extensors is repeatedly noted as the dominant clinical finding. Weakness of the hip flexors and sensory abnormalities along the anterior thigh and medial aspect of the lower leg are also commonly seen. Severe injuries may reveal a loss of the patellar reflex on the affected side.

Electrodiagnosis

Femoral nerve motor conduction studies have been described by Johnson et al.[569] In the electrodiagnostic laboratory Walter Reed Army Medical Center, recording electrodes are placed over the vastus medialis 32 cm distal to the inguinal ligament. The reference electrode is placed 4 cm distal to the active electrode. Stimulation below the inguinal ligament is 30 cm from the active electrode. Stimulation above the inguinal ligament is 35 cm from the active electrode. The ground electrode is placed between the stimulating and active electrodes. Abnormal latencies are greater than 7.5 ms and 8.4 ms below and above the inguinal ligament, respectively. Proximal, neuropraxic injuries following iliopsoas hematomas may not reveal significant findings on the distal motor-evoked potentials, although compression by the inguinal ligament may show a conduction delay across the segment. Sensory conductions can be obtained along the saphenous nerve. Stimulation occurs at the intersection of the medial gastrocnemius and the tibia. The active electrode is placed 14 cm from the stimulation point, between the medial malleolus and the tibialis anterior tendon. The reference electrode is placed 3 cm distal to the active electrode. Amplitudes less than 0.5 mV and latencies greater than 4.4 ms are greater than two standard deviations from the mean and are considered abnormal.[570] Saphenous nerve somatosensory-evoked potentials are likely the only helpful conduction study when a very proximal conduction block is suspected.[571] An EMG provides a more reliable assessment of femoral nerve pathology. Injuries proximal to the inguinal ligament will commonly involve the iliopsoas muscle. Variable abnormalities may be seen when the focus of injury lies from just proximal to the inguinal ligament to just above the femoral triangle, as many variations of motor branching to the quadriceps, pectineus, and sartorius muscles may occur in this region. However, at this relatively distal level, the iliopsoas muscle should be spared.

Treatment

Most reports suggest that conservative treatment is satisfactory in the majority of patients. Return to normal levels of function ranged from several months to a year. In the rare situation where retroperitoneal hemorrhage leads to progressive weakness, surgical exploration and decompression may be warranted.

Peroneal Nerve Injury

Anatomic Consideration

The common peroneal nerve fascicles incorporate the lateral portion of the sciatic nerve. At a variable level, just above the popliteal region, the sciatic nerve splits into its distinct branches, the common peroneal and tibial nerves (Figure 9-81). The common peroneal nerve courses laterally along the medial border of the biceps femoris muscle, crossing the proximal end of the lateral gastrocnemius muscle. The nerve becomes superficial as it passes between the fibular head and the peroneus longus muscle. Two branches diverge as the trunk passes anteriorly (Figure 9-82). The superficial peroneal nerve dives between the peroneus longus and the extensor digitorum longus and provides innerva-

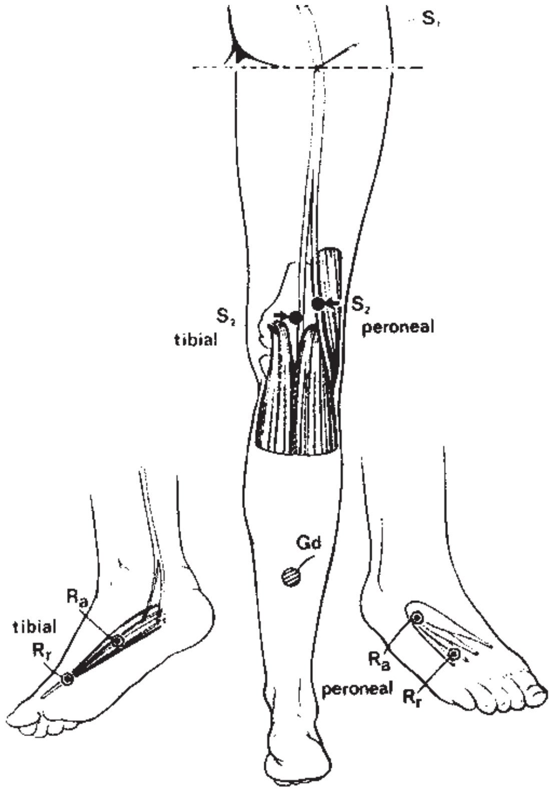


Fig. 9-81. The sciatic nerve dividing in to common peroneal and tibial branches above the popliteal fossa. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 201.

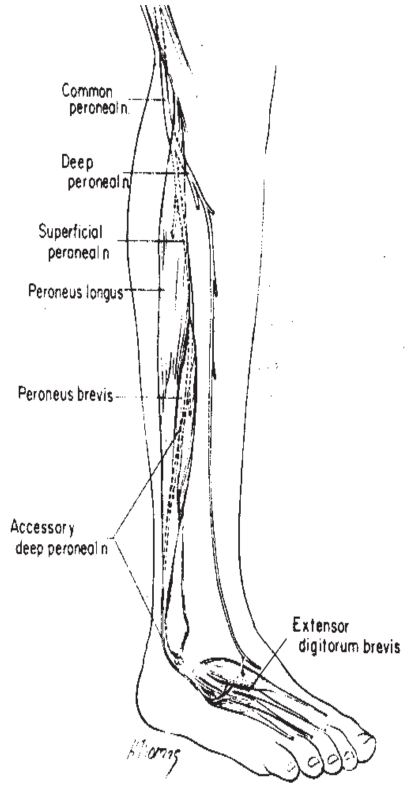


Fig. 9-82. Branches of the common peroneal nerve including the deep peroneal, superficial peroneal, and accessory deep peroneal nerves. Adapted with permission from Ma DM, Liveson JA. *Nerve Conduction Handbook.* Philadelphia, Pa: FA Davis Co; 1985: 208.

tion to the peroneus longus and brevis. The sensory portion continues distally, becoming superficial at the distal third of the leg. The superficial peroneal nerve transmits sensation from the anterolateral aspect of the distal half of the leg. Distal branches, including the intermediate and medial dorsal cutaneous nerves, supply sensation to the dorsum of the foot. Sensation along the first web space and the lateral border of the foot is provided by the deep peroneal and sural nerves, respectively, and not by the superficial peroneal nerve.

The deep peroneal nerve continues to run inferomedially along the medial aspect of the fibula, deep to the extensor digitorum longus muscle, anterior to the interosseus membrane. It provides articular branches to the knee and motor innervation to the tibialis anterior, extensor digitorum longus, and extensor hallicus longus muscles. The nerve dives beneath the extensor retinaculum between the extensor hallicus longus and tibialis anterior tendons. The lateral branch of the deep peroneal nerve innervates the extensor digitorum brevis and peroneus tertius muscles. The medial branch provides sensory innervation to the first web space.

Etiology

The course of the peroneal nerve makes it particularly susceptible to both compression and stretch injuries. As it passes beneath the head of the fibula, it lies intimately with the fibular periosteum for 10 cm. The initial 4 cm are covered only by skin and superficial fascia. Passage through a tunnel formed by the peroneus longus muscle, the fibula, and intermuscular septum limits the nerve's longitudinal excursion to merely 0.5 cm.[572] While entrapment within this tunnel is exceedingly rare, repetitive injuries may lead to adhesions, further limiting the mobility of the nerve (see Figure 9-82).

The vast majority of common peroneal nerve injuries occur at the nerve's most vulnerable point, at the fibular head. Mild injuries have been noted to occur spontaneously.[563,573] It is likely, however, that they developed secondary to inadvertent prolonged compression, possibly from limited movement during sleep, extended or repetitive leg crossing, or kneeling. Persons who have had an abrupt loss of weight are felt to be particularly susceptible, in light of the loss of protective superficial and epineural fat.[421] Ten percent of all sports-associated nerve injuries involve the peroneal nerve.[574] Minor trauma resulting in peroneal nerve injury during participation in sports including soccer, running, rugby, racquetball, mountain climbing, and bungee cord jumping have been anecdotally reported.[575–578] More severe injuries are associated with more profound trauma. Inversion injury at the ankle generates traction forces on the peroneal nerve at the fibular head. The Nitz et al[579] series revealed a 17% incidence of peroneal nerve injury with grade II lateral ankle sprains and an astonishing 86% incidence with grade III injuries. Trauma such as that sustained during motor vehicle accidents, which generate varus forces at the knee powerful enough to disrupt the lateral collateral ligaments, fracture the distal femur or proximal tibia, or dislocate the knee, commonly injure the peroneal nerve.[575,580]

Iatrogenic injuries to the peroneal nerve have also been documented. Application of casts and orthoses may compress the nerve against the fibular head. Prolonged compression due to poor positioning can occur during operative procedures. Injury following application of sequential pneumatic compression devices for deep vein thrombosis prophylaxis has been reported.[581]

Palsy of the deep peroneal branch is a frequent complication of external fixation during proximal tibial osteotomies, performed as treatment for severe varus deformities.[582] Limb lengthening through application of Ilizarov external fixation requires repeated elongation of 0.25 mm every 6 hours.[583] The tension-stress developed by distension of the fixator stimulates bone formation, a process described by Ilizarov as distraction osteogenesis.[584] Stretching of the soft tissues spanning the focus of lengthening is a potentially injurious corollary. A small series involving patients undergoing tibial lengthening revealed mild axonal sensorimotor peroneal neuropathies in all subjects.[585]

The ubiquitous use of arthroscopy in the diagnosis and treatment of knee injuries has led to a small but significant number of peroneal nerve injuries. The rate of serious neurological complications is low, less than 1% in several retrospective studies.[586,587] Rodeo et al[588] suggested that the position of the common peroneal nerve at the level of the joint line places it at risk during passage of large bore needles and especially by suture placement during lateral meniscal repair. Esselman and colleagues[589] caution that the topography of the common peroneal nerve at the level of the knee places the deep peroneal fascicles at greater risk than the superficial peroneal fibers. Great care needs to be taken during electrodiagnostic evaluations to prevent the erroneous diagnosis of a more distal lesion.[589]

Clinical Presentation

Weakness of the ankle dorsiflexors, especially the tibialis anterior muscle, is the most profound consequence of injury to the peroneal nerve. Incomplete paralysis impairs the ability of the dorsiflexors to contract eccentrically during heelstrike. The uncontrolled plantar flexion segue to footflat produces the foot-slap gait abnormality. Severe or complete paralysis forces the patient to adopt a steppage gait. Exaggerated hip and knee flexion is needed during the swing phase to clear the plantar flexed foot.[590] Weakness of the foot evertors and toe extensors are also commonly found on examination and may cause mediolateral instability during the stance phase. Sensory deficits were perceived by 79% of subjects in Katirji and Wilbourn's[591] study and variably involved the lateral aspect of the leg and dorsum of the foot.

Electrodiagnosis

Katirji and Wilbourn's series[591] of over 100 patients revealed four distinct patterns of peroneal nerve injury. Electrodiagnostic studies are essential in delineating these injuries, which prognostically are very different. They are also key tools in the differentiation of peroneal nerve injuries at the fibular head from more proximal problems including sciatic neuropathies, lumbar plexopathies, and L-5 radiculopathies.

The most important information can be gleaned from the peroneal motor NCS. Active electrodes are commonly placed on the extensor digitorum brevis muscle. Stimulation is applied just below the fibular head and at the popliteal fossa. In situations where a CMAP cannot be elicited from the extensor digitorum brevis muscle because of atrophy or local trauma, electrodes should be placed over the tibialis anterior muscle. Of the parameters assessed, the amplitude of the evoked potential is of the greatest utility. Decreased amplitudes above and below the fibular head suggest axonal loss, while normal amplitudes below the fibular head, but with decreased or absent amplitudes at the popliteal fossa, are more indicative of conduction block. Prolongation of conduction velocities across the fibular head of greater than 10 ms relative to more distal conduction velocities have been touted as significant. They have not, however, been shown to be clinically relevant in terms of dorsiflexor weakness or ultimate prognosis.[573,591,592]

Sensory nerve action potentials can be obtained by stimulation of the superficial peroneal nerve. The active electrode is positioned one finger breadth medial to the lateral malleolus. Stimulation is applied 12 cm proximal to the active electrode, along the medial border of the peroneus longus muscle and the edge of the fibula.[593] Again, the amplitude of the action potential provides the most significant information. Normal amplitudes in the face of foot drop suggest either a lesion proximal to the dorsal root, conduction block at the fibular head or, less commonly, isolated injury to the deep peroneal fascicles with sparing of the superficial fibers. Decreased amplitudes or inability to obtain a potential suggests axonal injury. Posterior tibial motor and sural sensory NCSs as well as H-reflexes should be performed to help differentiate more diffuse and proximal etiologies.

Electromyography is essential in the evaluation of peroneal neuropathies. Two muscles innervated by the deep branch should be tested, including the tibialis anterior and either the extensor hallucis or the extensor digitorum brevis. One superficial peroneal innervated muscle, either the peroneus longus or peroneus brevis should also be examined. Testing of muscles within the L-5 myotome but not in the peroneal distribution such as the tibialis posterior or flexor digitorum longus is helpful in determining the likelihood of more proximal injuries such as proximal sciatic neuropathies, lumbar plexopathies, and most importantly, L-5 radiculopathies. Electromyographic evaluation of the short head of the biceps femoris muscle should always be performed. Innervated by the peroneal portion of the sciatic nerve, it is the only peroneal-innervated muscle above the knee. Its involvement is exceedingly helpful in injury localization.

According to Wilbourn,[592] the most common presentation is axonal injury at the fibular head. Nerve conduction studies reveal low amplitude CMAP during stimulation at both the popliteal fossa and distal to the fibular head. Sensory nerve action potentials are either not obtainable or of very low amplitude. Electromyography helps to localize the lesion. Evidence of denervation observed in muscles innervated by the tibial nerve, containing fascicles arising from the L-5 nerve root is normal. Needle examination of the short head of the biceps muscle helps to determine whether the injury is distal to its branch site in the mid thigh. Denervation of the short head of the biceps suggests a more proximal lesion involving the peroneal portion of the sciatic nerve.

The second most common type of injury is focal conduction block at the fibular head. CMAPs distal to the fibular head are normal, while those attained by popliteal stimulation are either not obtained or are of decreased amplitude. Interestingly, incomplete conduction block in this situation rarely causes temporal dispersion. Sensory action potentials are normal. Electromyographic findings are of little help and may in fact be confusing if not considered in relation to the NCSs. An injury severe enough to cause complete conduction block frequently injures at least a small number of axons. Sparse evidence of denervation may be seen in several muscles. It is important to realize that the discrete interference pattern and minimally increased recruitment frequency are products of focal conduction block and not severe axon loss.

A relatively rare presentation is a mixed axon loss-conduction block injury. Motor conduction studies reveal decreased action potential amplitudes below the fibular head and either absent or more severe attenuation of the amplitude during stimulation at the popliteal fossa. SNAPs are absent. Electromyography suggests significant denervation of muscles innervated by the deep and superficial peroneal branches.

Isolated axonal injury of the deep peroneal fascicles exclusively is extremely unusual. Proximal and distal CMUAP are of low amplitude or not obtainable. The superficial peroneal sensory potential is normal. Sensory NCSs of the deep peroneal nerve may be helpful.[594] While technically difficult, assymetry between affected and nonaffected limbs may suggest a focus distal to the dorsal root ganglion. Electromyography is important in this situation. Denervation will be observed just within muscles innervated by the deep peroneal nerve.

Treatment

Initial electrodiagnostic studies are of value in predicting the outcome of peroneal palsies and may help guide treatment plans. Berry and Richardson[572] observed a correlation between persistent, moderate sensorimotor deficits and initial distal motor conduction velocities of less than 30 meters per second. Smith and Trojaborg[573] also observed that normal sensory and motor conduction parameters below the fibular head invariably lead to a good prognosis. Abnormal distal latencies or significantly decreased amplitudes were associated with incomplete recovery. Wilbourn's[592] findings parallel those of the previous researchers. Focal conduction block at the fibular head typically resolved completely, within 8 to 12 weeks. Axon injury at the fibular head resulted in satisfactory but incomplete recovery and usually took upward of 6 months. More proximal axon loss such as those exhibited in sciatic neuropathies and L-5 radiculopathies portended poor outcomes. Mixed axon loss-conduction block injuries recovered in a bimodal fashion, with the conduction block resolving in weeks and reinnervation occurring several months later. Incomplete functional return was common.

Treatment of peroneal neuropathies varies with the severity of the injury. Maintenance of full range-of-motion at the ankle is of paramount importance. Prolonged stretching of the gastrocnemius-soleus complex should be performed several times a day. Progressive resistance strengthening should also be started early and include not only the dorsiflexors but the foot everters as well. Proprioceptive retraining at the ankle should be instituted prior to return to normal activities. Ankle foot orthoses are not necessary in situations where adequate toe clearance can be achieved. In cases of severe weakness of the foot dorsiflexors, moderate dorsiflexor weakness but with rapid fatigue or mediolateral instability, an ankle foot orthosis is indicated. An exhaustive discussion of this subject can be found in chapter 11, *Orthotics for the Injured Soldier.*

NERVE INJURIES OF THE FOOT AND ANKLE

Entrapment neuropathies of the foot and ankle are uncommon. The actual incidence is unknown.[595] They include entrapment of the superficial peroneal, sural, deep peroneal, and posterior tibial nerve or its branches, the medial and lateral plantar nerves, the calcaneal nerve, and the interdigital nerves. Entrapment of the deep peroneal nerve is also known as the anterior tarsal tunnel syndrome. Entrapment of the posterior tibial nerve or its branches is typically referred to as the tarsal tunnel syndrome. It is also referred to by some as the posterior tarsal tunnel syndrome. The term *entrapment* in this discussion will include those nerve injuries due to direct trauma as well as chronic compression.

These nerve injuries are likely underdiagnosed.[595,596] There may be a number of reasons for this situation. First, many patients present with a history of vague or fleeting symptoms, initially accompanied by only minimal or no definite neurologic deficits. If the patient presents to coaches or trainers, he may find that they are typically much more aware of soft-tissue injuries of the foot and ankle than they are of nerve injuries; thus, the index of suspicion may be relatively low, particularly on initial evaluation. This lack of awareness regarding the types and nature of entrapment neuropathies of the foot and ankle may also hold true for many physicians and podiatrists not experienced in this type of injury. The neural problems often coexist with soft-tissue injuries. The latter conditions typically receive initial treatment while the nerve injury either receives delayed attention or goes unnoticed altogether. In addition, the signs and symptoms of nerve injury may be temporally related to a given activity for only a brief period of time immediately following the activity. Thus, the nerve insult may not be appreciated when the individual is assessed well after the precipitating activity. Finally, the diagnosis may be difficult to detect by objective findings. Electrodiagnostic studies, including both NCSs and needle EMG, can be particularly helpful in confirming and localizing the site of nerve entrapment and may also be helpful in assessing the severity of the injury while offering clues regarding the prognosis of the nerve lesion. However, electrodiagnostic studies are primarily an extension of the physical examination and may not be sensitive enough to pick up subtle or transient nerve pathology. In addition, they require appropriate timing to obtain meaningful information for accurate diagnosis and prognostication.

Although the number of neurologic injuries at the foot and ankle are undoubtedly a small percentage of the total, it is worthwhile examining the type and percentage of acute and overuse injuries of the foot and ankle, since they appear to play a role in causing nerve damage. Garrick and Requa[597] assessed 16,754 injuries occurring in a variety of sports and found that one quarter of the injuries were to the foot and ankle; 9.7% involved the ankle, 15.5% the foot.

They noted that over half (50.4%) of the ankle injuries were acute sprains. Volleyball and basketball had the highest proportion of acute ankle sprains at 82% and 79% of participants, respectively. Football and racquetball each had more than 70%.[597]

The highest proportion of overuse injuries of the ankle occurred in cycling (70%); ice skating (40.5%); and ballet and running, each with more than 35%.

Volleyball had the highest percentage of overuse injury of the foot at 70%; followed by running at 59%; and then by tennis, gymnastics, racquetball, and skiing, each at 50% or above.[597]

According to Schon and Baxter,[596] neurologic injuries of the foot and ankle occur most commonly in runners or joggers (60%) and dancers (20%). They add that the most common neurologic problems in athletes are interdigital neuromas, followed in descending order by entrapment of the first branch of the lateral plantar nerve, medial plantar nerve, tibial nerve, lateral plantar nerve, deep peroneal nerve, superficial peroneal nerve, and sural nerve.

Tarsal Tunnel Syndrome

Posterior tarsal tunnel syndrome is perhaps the best known of the entrapment neuropathies of the foot and ankle. Tarsal tunnel syndrome is defined as the entrapment of the tibial nerve or one of its branches by the flexor retinaculum. Kopell and Thompson[598] were credited with the first description of this nerve injury in 1960. Keck and Lam, however, were independently the first to coin the term tarsal tunnel syndrome (as cited by Radin[599]).

Anatomic Considerations

The tarsal tunnel is formed by fibrous and osseous structures, including the flexor retinaculum or laciniate ligament, which forms the roof. The medial wall of the calcaneus, the posterior aspect of the talus, the distal tibia, and the medial malleolus complete the tunnel. The tendinous arch of the abductor hallucis muscle contributes to the medial wall of the tunnel along with the laciniate ligament.[600] The latter ligament has both a superficial and a deep layer. The superficial layer is a thickening of the crural fascia between the medial malleolus and the calcaneal tuberosity and the deep layer extends from the medial malleolus to insert on the crural fascia. The deep layer lies over the sustentaculum tali and posterior talar process (Figure 9-83).[600]

The tibial nerve, the posterior tibial artery and vein, and tendons of the posterior tibialis, flexor digitorum longus, and flexor hallucis longus all pass through the tunnel. The posterior tibial nerve typically divides 1 to 2 cm proximal to an imaginary line drawn from the tip of the medial malleolus to the calcaneal tuberosity. The medial and lateral plantar nerves, branches of the tibial nerve, each then enter one of the two tunnels located within the abductor hallucis muscle.

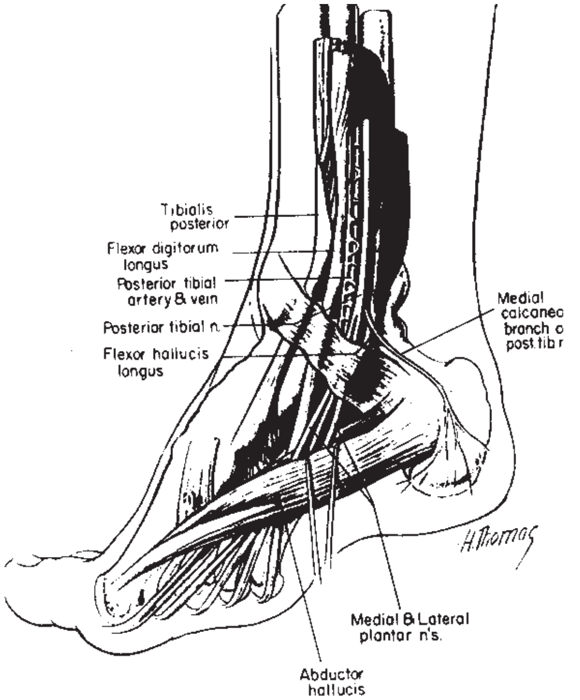


Fig. 9-83. The tarsal tunnel. The tibialis posterior, flexor digitorum longus, flexor hallucis longus, posterior tibial nerve, artery, and vein pass beneath the flexor retinaculum. Reprinted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 54.

The medial plantar nerve supplies sensation to the medial aspect of the plantar surface of the foot and the medial half of the fourth toe. It also provides a motor branch to the abductor hallucis, the short toe flexors, and the first lumbrical muscle.

The lateral plantar nerve provides sensation to the fifth toe and the lateral half of the fourth toe. It also supplies sensory innervation to the lateral sole and motor fibers to the remaining intrinsic muscles of the foot, except for the short toe extensor. As previously noted, the medial and lateral plantar branches pass via separate openings in the medial superior origin of the abductor hallucis muscle. This anatomic structure predisposes these nerves to selective entrapment.[601] The first branch of the lateral plantar nerve passes obliquely between the abductor hallucis muscle and the quadratus plantae. It subsequently divides into three branches. One innervates the periosteum of the medial process of the calcaneal tuberosity; it also frequently innervates the long plantar ligament and occasionally the quadratus plantae muscle. Another supplies the flexor digitorum brevis muscle. The terminal branch innervates the abductor digiti minimi muscle.[596]

The calcaneal nerve branch lies posterior to the aforementioned branches and may pierce the laciniate ligament or exit between it and the fascia of the abductor hallucis muscle. Branches of the calcaneal nerve piercing the laciniate ligament innervate the medial aspect of the calcaneus, while other branches travel the length of the tarsal tunnel to innervate the plantar aspect of the heel.[601] Just proximal to the tarsal tunnel, a calcaneus branch splits from the tibial nerve to innervate the skin of the heel. This branch is susceptible to nerve compression at the edge of the laciniate ligament. If calcaneal nerve entrapment does occur, a patient typically complains of heel pain.[602]

Etiology

Tarsal tunnel syndrome has been attributed to a multitude of etiologies. Increased pressure within the tarsal tunnel is felt to be the common denominator in causing nerve injury. This may occur as the result of direct compression injury. It may also result from decreased volume due to space-occupying changes within this nonyielding, fibroosseous tunnel. The synovial proliferation associated with tenosynovitis; soft-tissue masses such as ganglion, lipomas, neurilemomas, and neurofibromas, as well as local bleeding; and venous engorgement, edema, or both, are space-occupying conditions that may cause focal nerve injury within the tunnel.[600]

Bony anomalies may cause compression neuropathy. Schon and Baxter[596] report two cases in which bony abnormalities of the posterior talus caused such nerve compression. Metabolic disorders or hormonal changes, such as those that occur with diabetes, pregnancy, myxedema, acromegaly, and hyperlipidemia, predispose an individual to compression neuropathy.[600]

The medial plantar neurovascular structures pass through the upper section of the tarsal tunnel. This upper section is reportedly narrower than the lower and accounts for the suggestion that the medial plantar vessels and nerves are more sensitive to volume changes than the lateral plantar neurovascular structures which pass through the lower section.[600] Jackson and Haglund[595] state, however, that entrapment of the tibial nerve most commonly occurs at the anterior, inferior aspect of the tarsal tunnel at the point the nerves wind around the medial malleolus. They agree with Kaplan and Kernahan[603] that the lateral plantar nerve branch is more frequently subject to compression neuropathy than the medial branch.[595]

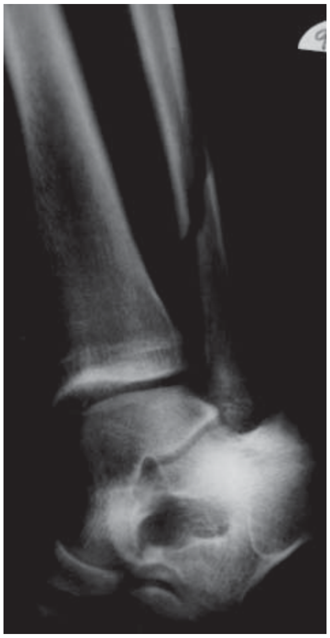


Fig. 9-84. Pronation–external rotation injury at the ankle, resulting in tibial nerve injury, rupture of the anterior talofibular ligament, incomplete tear of the deltoid ligament, transverse fracture of the medial maleolus, spiral fracture of the distal fibula, and fracture of the posterior lip of the distal tibia. Radiograph courtesy of MAJ Aron M. Judkiewicz, M.D., Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

The majority of nerve injuries involving the tarsal tunnel have been recorded as posttraumatic complications.[422] Many of these have been considered to be the result of scar formation after an ankle sprain and others have been thought to be due to secondary osseous changes. Ankle sprains involving the deltoid ligament and calcaneal or medial malleolus fractures or both have been cited as possible causes (Figure 9-84).[604]

Kraft[605] notes that the tibial nerve is especially vulnerable during ambulation because of its location and the structure of the tarsal tunnel. He feels that the distal branches of the tibial nerve are sometimes injured as a result of repetitive trauma rather than chronic, steady compression. The repetitive action at the ankle experienced with running, especially when associated with excessive pronation, is thought to place the tibial nerve "on stretch" on a repetitive basis.[595] Rask[606] reports that excessive valgus or external rotation of the foot, particularly that experienced with running, potentially causes excessive stretch of the medial plantar nerve and referred to this problem as "jogger's foot." The fact that prolonged standing or walking typically precipitates or exacerbates symptoms supports the contention that fluid stasis or engorgement within the tarsal tunnel is a factor in causing compression.[422]

Other suspected causes or predisposing conditions include inflammatory soft tissue conditions associated with rheumatoid arthritis, ankylosing spondylitis, Reiter's disease, collagen vascular diseases, and isolated flexor tenosynovitis secondary to acute or chronic injury.[596,604] Chronic thrombophlebitis has also been included in the list of potential causes.[422,604] Ricciardi-Pollini et al[607] reported eight cases of tarsal tunnel syndrome and found a thickened laciniate ligament, varices, exostoses, and adhesions to be causative factors. Jackson and Haglund[595] add that gout with urate deposits, connective tissue changes associated with aging, as well as fluid retention and bone spurs may also have a role in exacerbating or precipitating nerve injury.

Some have implicated severe pronation of the hindfoot as a potential cause.[596] DeLisa and Saeed[604] summarized multiple reports in the literature that indicate that abnormal calcaneal eversion relative to an adducted talus and an abducted forefoot may place increased tension on the neurovascular structures within the tunnel and cause this syndrome. Hypertrophy of the abductor hallucis muscle or an accessory muscle belly of the short digital flexors, rapid weight gain, and constant squatting are additional causes reported in the literature.[595,604]

Fewer cases have been noted as the consequence of benign tumors, such as neurilemomas, ganglion, and lipomas. Others have been documented as the result of varicosities beneath the flexor retinaculum.[602]

Jackson and Haglund[595] reported two selected cases of tarsal tunnel syndrome. One occurred as the result of direct, extrinsic compression attributable to a tight alpine ski boot. The other occurred in a female runner with bilateral plano valgus deformities and increased Q-angles.

Schon and Baxter[596] purport two mechanisms for tarsal tunnel syndrome. They include vascular compromise of the nerve by pressure on the vasa nervorum and direct compression neuropathy. These authors feel that the former mechanism results in primary sensory deficits without motor symptoms or signs while the latter results in both sensory and motor signs and symptoms. Pecina and colleagues[600] note that the tibial nerve, though rich in vascularity, is sensitive to ischemic insult and agree that compression of the vasa nervorum and the resulting ischemia may manifest as neurologic symptoms consistent with tarsal tunnel syndrome.

Clinical Presentation

Patients typically present with complaints of numbness and tingling involving the toes and plantar aspect of the foot. Onset is usually insidious.[599] The distribution of sensory involvement is quite variable, reflecting the variable involvement of the tibial nerve or its branches. Sensory distribution of the medial or lateral plantar nerves or both are characteristically involved.[598] Individuals may also complain of burning pain and dysesthesias in a similar distribution. At times, particularly with prolonged activity, symptoms may be relatively constant with activity-induced exacerbations. In general, however, symptoms are intermittent and precipitated or exacerbated by prolonged standing, walking, running, or similar activities involving dynamic action at the foot and ankle. Rest usually diminishes such symptoms. Nocturnal paresthesias and pain are common and may be severe enough to prohibit sleep or, more typically, awaken the patient from sleep.[604] Not infrequently, patients complain of pain radiating up the medial aspect of the calf and this presentation may lead one to erroneously presume the presence of a radiculopathy.[600]

Sensory impairment classically involves the sole of the foot. Anesthesia and hyperesthesia are reportedly rare, while hypesthesia and loss of two-point discrimination are early signs of nerve compression.[600] A positive Tinel's sign may be elicited over the tarsal tunnel site and passive placement of the heel into an end-range valgus and everted posture[604] or eversion with dorsiflexion at the ankle[600] may exacerbate symptoms. The Valleix phenomenon, the situation where a Tinel's sign is obtainable in the tarsal tunnel region and tenderness is elicited both proximal and distal to the compression site, may be demonstrated on occasion.[608] Passive varus alignment of the heel, on the other hand, typically reduces the symptoms presumably due to creation of "slack" in the laciniate ligament.[604] There is often tenderness with palpation at a point posterior and inferior to the medial malleolus. There is, however, a lack of tenderness with palpation in the area of the sole of the foot where the patient is typically most symptomatic.[599] A variable degree of retro- or submalleolar swelling may be noted upon inspection.[600] If motor impairment is present, it usually manifests as weakness of the toe flexion at the metatarsophalangeal joints and toe extension at the IP joints of all or some of the toes.[604] In contrast, strength of the long flexors of the foot and toes is preserved.

Differential Diagnosis

The differential diagnosis should include Achilles tendinitis, plantar fasciitis, prolapsed metatarsal heads, plantar callosities, longitudinal arch sprain, localized rheumatoid disease, other arthritic conditions, sciatica, peripheral neuropathy, peripheral vascular disease, old fractures, bone spicules, accessory ossicles, and an S-1 radiculopathy.[595] Diabetic neuropathy with burning paresthesias of both feet may be particularly difficult to distinguish from bilateral tarsal tunnel syndrome by clinical exam alone.[422]

Radiographic and electrodiagnostic studies are of potential value in the evaluation for tarsal tunnel syndrome. Radiograph examination of the ankle may detect the presence of bony anomalies, ankle fractures, accessory ossicles, and ankle malalignment. These abnormalities may be factors in precipitating the syndrome or any one may be the primary source of the symptoms. Careful clinical evaluation will assist in making this distinction. Ricciardi-Pollini et al[607] documented a case involving a young woman who was noted by radiograph to have an exostosis located at the medial site of the talus. At surgery to remove this osseous lesion, it was noted to be compressing the posterior tibial nerve. Its removal, coupled with neurolysis, resulted in complete relief of her symptoms.

Electrodiagnosis

Electrodiagnostic studies may be of particular importance not only in reliably diagnosing a peripheral neurologic deficit, but in providing information regarding the severity and specific distribution of nerve involvement. Such studies may be particularly helpful in distinguishing a peripheral neuropathy from a bilateral tarsal tunnel syndrome.

Multiple studies are cited by DeLisa and Saeed[604] which demonstrate prolonged sensory or motor distal latencies, or both, across the tarsal tunnel region. These authors found the mixed nerve conduction technique as described by Saeed and Gatens[609] substantially superior to the medial and lateral plantar orthodromic sensory nerve conduction technique employing a ring electrode stimulator at the great toe and fifth toe, respectively. The recording electrode in the latter case is located just proximal to the flexor retinaculum. This method can be difficult to perform technically, particularly because one typically requires an averager to obtain an evoked potential of acceptable amplitude. On the other hand, the mixed nerve conduction technique involves orthodromic stimulation of the medial or lateral plantar nerves in the midsole of the foot. This stimulation is done at a predetermined distance with the recording electrode located proximal to the flexor retinaculum. This technique is typically reliable and reproducible.

Motor NCSs through the tarsal tunnel are an additional means of assessing the integrity of the medial or lateral plantar nerves. In the case of the medial plantar nerve study, the active pickup electrode is located over the motor point of the abductor hallucis muscle. The lateral plantar nerve motor NCS is performed with the active pickup electrode placed over the abductor digiti minimi. In both cases, the site of stimulation is 10 cm proximal to recording electrodes at a point just proximal to the flexor retinaculum.[604]

Needle electrode study of foot intrinsic muscles served by either the medial or lateral plantar nerves may detect electrodiagnostic abnormalities consistent with muscle membrane instability and suggestive of a denervation process. DeLisa and Saeed[604] considers the abductor hallucis and the first dorsal interosseus muscles especially useful in the initial screening process.

In the presence of tarsal tunnel syndrome, one will typically obtain either prolonged or absent sensory or CMAP. In addition, there may be evidence of denervation activity, along with reduced recruitment and interference patterns involving the foot intrinsics.

Treatment

Conservative management is the initial treatment of choice. A trial of an NSAID and custom-molded orthotics are typically offered. Injection of the tarsal tunnel site with a mixture of a local anesthetic and corticosteroid compound may be performed for both diagnostic and therapeutic purposes. Pecina and colleagues[600] feel repeat corticosteroid injections may be performed at the same site up to three times over a two-month period. If unsuccessful and no other conservative measure offers relief, they then feel a surgical option should be entertained. Ice and ultrasound have also been employed with variable success.

Appropriate alteration in footwear may be warranted for improved ankle and foot stability. A custom-molded foot orthotic may be added to provide optimal heel stability and prevention of excessive forefoot pronation. Use of a medial arch support as well as an external heel counter on the shoe are two additional means of achieving appropriate foot alignment. Rask,[606] however, notes that arch supports should be avoided because they may cause compression injury to the medial plantar nerve in the region of the longitudinal arch. The latter issue is controversial and will receive further elaboration later in the discussion of medial plantar entrapment neuropathy in the region of the longitudinal arch.

Relative rest with elimination of the suspected precipitating or exacerbating activities is a particularly important measure one should consider. This especially includes elimination of repetitive trauma in the region of the tarsal tunnel, as well as repetitive or marked stretch of the tarsal tunnel elements. Complete immobilization has not been required in this author's experience, although some advocate short-term use of plaster casts.[600] Pecina and colleagues[600] reported a study by Androic that purportedly documented success as high as 79% with conservative measures.

For refractory cases, to include those patients suffering from tarsal tunnel syndrome as the result of varices, exostoses, and adhesions, surgical decompression along with neurolysis may be warranted. Ricciardi-Pollini and colleagues[607] reported a small series of eight patients undergoing surgical decompression due to the latter problems as well as to a thickened laciniate ligament. All but one received complete relief of their symptoms within 24 hours after surgery. Radin[599] studied a series of 14 patients with documented tarsal tunnel syndrome with associated varus heels and pronated, splayed feet. He argued that surgical release of the flexor retinaculum posterior to the medial malleolus is the treatment of choice in individuals with tarsal tunnel syndrome and a planovarus deformity. He reported successful outcomes with surgical intervention in more than 90% of the cases. Kaplan and Kernahan[603] surgically managed 18 patients with tarsal tunnel syndrome and reported complete or partial improvement in all.

Lateral Plantar Nerve Branch-I Entrapment

Entrapment of the first branch of the lateral plantar nerve should be considered in individuals suffering from chronic heel pain. This appears especially applicable for the athletic population. Schon and Baxter[596] report that about 10% to 15% of athletes with chronic, persistent heel pain have entrapment neuropathy of this particular nerve branch. Pecina and colleagues[600] note that runners and joggers make up the great majority of reported cases, but acknowledge that athletes in soccer, dance, and tennis also experience this problem.

Etiology

The site of entrapment is purported to occur between the deep fascia of the abductor hallucis muscle and the medial caudal margin of the head of the quadratus plantae muscle.[596,600] Chronic repetitive trauma at the latter site is the presumed cause. Local inflammation secondary to chronic pressure reportedly may also occur where the nerve passes over the plantar side of the long plantar ligament or in the osteomuscular canal between the calcaneus and the flexor digitorum brevis.[596] A review of the literature by Schon and Baxter[596] indicates that a hyperpronated foot, hypertrophied abductor hallucis or quadratus plantae muscle, accessory muscles, abnormal bursae, and phlebitis in the calcaneal venous plexus may all play a role in precipitating this nerve entrapment.

Clinical Presentation

Chronic heel pain is the typical presentation. Characteristically, the patient is an athlete involved in a sport requiring a running activity. Pain is typically exacerbated by the running activity or even by walking and is variably relieved with rest. Although radiation of pain to the ankle is not uncommon, numbness in the heel or foot is atypical.[596,600]

Reproducible point tenderness along the course of the nerve, particularly at the site of suspected entrapment, is present. This site is deep to the abductor hallucis muscle. A Tinel's sign may be elicited, but is reportedly an atypical finding.

Differential Diagnosis

Achilles tendinitis, plantar fasciitis, and bursitis should be included in the differential diagnosis. A roentgenogram should be obtained to exclude the possibility of heel spurs, stress fractures, and bone tumors. Occasionally, a bone scan may be warranted to clarify the diagnosis. Less common causes of heel pain that merit consideration include heterotopic calcification, Paget's disease, Strumpell-Marie disease, venereal disease, Sever's apophysitis, rheumatoid arthritis, and gout.[602] More proximal involvement of the tibial nerve, sciatic nerve, and an S-1 radiculopathy should be excluded. Electrodiagnostic testing, coupled with the history and physical examination, may prove helpful in ruling out the latter possibilities.

Treatment

Patients typically receive a trial of conservative therapy similar to that provided for heel pain due to other causes. This usually includes a trial of an NSAID, relative rest, ultrasound, stretching, heel cups, heel doughnut pads, and cortisone injections with local anesthetic. The latter treatment regime is sometimes successful. However, in refractory cases, typically those that persist after 6 to 12 months of conservative therapy, surgical release of the first branch of the lateral plantar nerve is indicated.[600]

Medial Plantar Nerve Entrapment at the Longitudinal Arch

Isolated entrapment of the medial plantar nerve in the longitudinal arch is a rarely diagnosed condition. Rask described such an entrapment neuropathy in joggers, and as previously mentioned, coined this clinical syndrome the "jogger's foot."[606]

Anatomy

The course of the medial plantar nerve has been previously discussed.

Etiology

The entrapment of the medial plantar nerve in this particular syndrome is felt to occur in the region of the longitudinal arch, also known as the master knot of Henry.[600,606] It has been attributed to chronic, focal inflammation due to repetitive trauma in the latter area. Rask[606] contends that long-distance running is the typical precipitating activity, particularly if excessive valgus displacement and external rotation occurs with this activity. He feels the latter presentation causes excessive stretch of the medial plantar nerve against the fibromuscular tunnel through which it passes.

Clinical Presentation

The typical patient is a middle-aged individual who jogs on a regular basis.[600] Rask[606] notes that chronic burning pain in the heel region is the typical presentation. Pecina and colleagues[600] note that onset of discomfort is characteristically temporally related to using a new arch support.

Impaired sensation involving the medial aspect of the sole of the forefoot and point tenderness at the site of entrapment in the arch of the foot, posterior to the navicular tuberosity, is characteristic.[600,606] A Tinel's sign may also be elicited.

Differential Diagnosis

More proximal entrapment of the medial plantar nerve in the tarsal tunnel, as well as tibial or sciatic nerve pathology, S-1 radiculopathy, and peripheral neuropathy should be excluded. Electrodiagnostic assessment, performed as an extension of the history and examination, usually permits one to confidently exclude these possibilities. Local anesthetic nerve block at the site of presumed entrapment typically results in temporary relief of symptoms and arguably excludes the possibility of calcaneal bursitis and plantar fasciitis.[606]

Treatment

Pecina et al[600]and Rask[606] note that this form of nerve entrapment typically responds to conservative management alone. A trial of relative rest, NSAID, and local anesthetic and cortisone injections have been implemented with some success. The injections are performed at the site of presumed entrapment just posterior to the navicular tuberosity and, as previously alluded, may have both diagnostic and therapeutic benefits.[606]

Rask[606] also advocates having the jogger modify his running style, specifically avoiding excessive valgus and external rotation when running. The jogger is encouraged to run on the lateral aspect of his foot with a very slight toe-in sprint in order to relieve pressure from the medial plantar nerve. As previously noted, Rask feels that arch supports should be avoided in joggers who develop medial plantar nerve entrapment neuropathy in the longitudinal arch area. He believes that such supports can cause further trauma to the medial plantar nerve in the latter region.[606] If the arch support is fabricated so that it is too high or causes focal pressure in the longitudinal arch, then his recommendation appears reasonable. However, if the arch support is custom-designed to accommodate the individual's arch and to evenly distribute pressure over a broad rather than focal area, then direct pressure trauma is likely to be minimal. In addition, the benefit derived in preventing hyperpronation of the forefoot and in stabilizing the foot and ankle arguably outweigh the likelihood of direct pressure trauma to the nerve.

In refractory cases, surgical neurolysis is advocated.[606] However, literature regarding the efficacy of surgical intervention for this particular form of entrapment neuropathy is lacking.

Anterior Tarsal Tunnel Syndrome

This syndrome is described as an entrapment of the deep peroneal nerve beneath the inferior extensor retinaculum. There has been a paucity of reports regarding this form of entrapment in the literature to date.[610–612] Kopell and Thompson[598] have been credited with identifying the syndrome in 1963.

This syndrome has been documented most commonly in runners. Other sports in which this problem has been identified include soccer, skiing, and dancing.[596]

Anatomic Considerations

In the proximal third of the leg, the deep peroneal nerve passes between the extensor digitorum longus and tibialis anterior muscles. In the middle third, it passes deep to the extensor hallucis longus muscle and tendon. At a point 3 to 5 cm above the ankle joint, the nerve courses between the extensor digitorum longus and extensor hallucis longus muscles. At 1 cm above the ankle joint, underneath the oblique superior medial band of the inferior extensor retinaculum, a branch to the extensor digitorum brevis emerges laterally. The medial branch of the deep peroneal nerve passes under the oblique inferior medial band of the inferior extensor retinaculum along with the dorsalis pedis artery. At this point the deep peroneal is susceptible to compression injury between the talonavicular joint ridges and the retinaculum (Figure 9-85).[596]

The inferior extensor retinaculum typically has three branches and forms a Y-shaped pattern transversely across the dorsum of the foot. The deep peroneal nerve has branches innervating all foot extensors except for the extensor digitorum brevis at the level of the anterior tarsal tunnel. This accounts for the fact that the only muscle typically observed to be involved in anterior tarsal tunnel syndrome is the extensor digitorum brevis.[600] The deep peroneal nerve also innervates the first dorsal interosseous muscle, but objective determination of atrophy and weakness involving this muscle is obviously difficult to assess accurately.[422]

In its distal course, the deep peroneal nerve pierces the dorsal aponeurosis of the foot where it becomes superficial. It then provides cutaneous innervation to the first web space, along with sensory supply to the lateral aspect of the great toe and medial aspect of the second toe.[596] A number of variations in the pattern of nerve branches off the deep peroneal nerve have been documented and are reportedly not uncommon.[610]

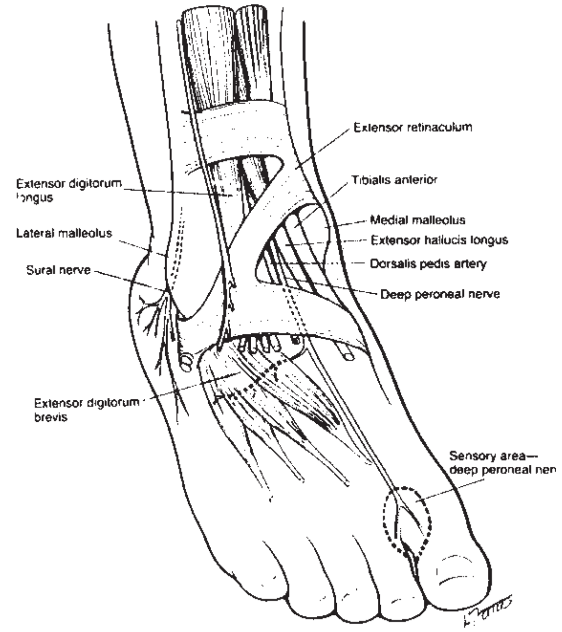


Fig. 9-85. The anterior tarsal tunnel. Branches of the deep and superficial peroneal nerve; tendons of the tibialis anterior and extensors digitorum longus and hallucis longus pass beneath the superior, oblique, and inferior bands of the extensor retinaculum. Adapted with permission from Liveson JA. *Peripheral Neurology: Case Studies in Electrodiagnosis.* 2nd ed. Philadelphia, Pa: FA Davis Co; 1991: 53.

Etiology

Anterior tarsal tunnel syndrome is usually attributed to chronic or repetitive compression injury at the ankle. Pecina and colleagues[600] contend that anatomical factors, specifically a tight, unyielding retinaculum overlying a bony structure, predisposes the deep peroneal nerve to such injury. Ankle edema; fractures; subluxations and sprains; as well as osteophytes, synovial pseudocysts, neuromas, ganglia, aneurysms, and tenosynovitis have been implicated as possible causes.[600] Bony anomalies of the talonavicular joint; repetitive compressive trauma from tight shoes, high boots, or shoe straps; chronic stretch injury due to prolonged ankle plantar flexion while wearing high heels; and direct ankle trauma have also been described as potential causes.[422,610] Maximal stretch of the deep peroneal nerve at the ankle has been documented to occur when end-range plantar flexion is coupled with dorsiflexion of the toes.[422] Schon and Baxter[596] cite from their experience that most patients have a history of recurrent ankle sprains, specifically those sprains in which the ankle is forced into plantar flexion and supination. They also note that they have observed this syndrome to occur in joggers who wear keys under the tongue of their shoes when running and soccer players who receive direct, often repetitive trauma to the dorsum of the foot. They additionally suggest that the deep peroneal nerve may sustain repetitive trauma in those individuals who perform sit-ups with their feet hooked under a hard surface.

Clinical Presentation

The typical patient complains of burning pain in the region of the web space between the great toe and second toe. There may also be subjective complaints of numbness in the latter area. The patient does not typically complain of any focal weakness. Nocturnal pain and paresthesias of the foot are not uncommon. Patients may point out that prolonged standing or walking, tight-fitting shoes, or walking in high heels precipitates or exacerbates their symptoms, while extension or eversion of ankle relieves their symptoms to some degree.[422,600]

Decreased sensation to light touch and pinprick is typically noted in the web space between the great toe and second toe. A Tinel's sign may be elicited with a tap over the anterior tarsal tunnel site. There may be also be variable weakness and atrophy of the extensor digitorum brevis muscle. The latter muscle is best tested with the ankle maximally dorsiflexed to eliminate the actions of the extensor hallucis longus and extensor digitorum longus muscles.[600]

Differential Diagnosis

The primary conditions which must be differentiated from anterior tarsal tunnel syndrome include more proximal peroneal nerve compression injury, especially at the fibular head, and an L-5 radiculopathy. If the lateral branch of the distal deep peroneal nerve is selectively injured, then sensory impairment will not be present. Instead, a patient may simply complain of foot pain. Under such circumstances, it is difficult to definitely exclude the possibility of arthritic pathology or ligamentous injuries in the ankle region. Focal atrophy involving the extensor digitorum muscle is one clue that helps to make this distinction. If more proximal peroneal nerve injury is present, then delineation of muscle weakness and atrophy above the level of the ankle may be present.[422]

Electrodiagnosis

Electrodiagnostic studies remain particularly helpful in excluding the possibility of more proximal peroneal nerve injury and an L-5 radiculopathy. A prolonged distal latency when stimulating at the ankle and recording at the extensor digitorum brevis, along with normal proximal conduction velocity, supports the presence of the anterior tarsal tunnel syndrome. In addition, needle electrode examination of the extensor digitorum muscle may yield evidence of muscle membrane instability suggestive of denervation activity, while sparing other lower extremities typically served by the deep peroneal nerve. It should be noted, however, that needle electrode abnormalities suggestive of denervation activity may be found in a small percentage of patients who are asymptomatic or without nerve conduction deficits or both, presumably due to the local muscle trauma that occurs with regular shoewear. Gessini and colleagues[610] reported four cases of anterior tarsal tunnel syndrome. Three of the four demonstrated significantly prolonged motor distal latency and the fourth was at the upper limits of normal. In addition, all four patients had needle electrode findings of denervation activity isolated to the extensor digitorum brevis muscle.

Roentgenograms of the ankle and foot may be helpful in excluding osseous lesions that may potentially compress the deep peroneal nerve, such as an osteophyte on the dorsum of the talus where it articulates with the navicular bone.[596]

Treatment

Conservative management is typically advocated and includes avoidance of constricting compression in the anterior tarsal tunnel region by tight shoes, boots, or shoelaces. Also, avoidance of high heels may eliminate the posture of ankle plantar flexion and toe dorsiflexion, a position felt to precipitate or exacerbate the symptoms of anterior tarsal tunnel syndrome. Use of shin guards with a lip over the dorsum of the foot when playing soccer may assist in preventing direct trauma to the anterior ankle. Relative rest and use of NSAIDs, local anesthetic, and corticosteroid injections may provide a variable degree of relief. Use of orthotics to maintain the ankle at 90° has been suggested, although there is no convincing evidence that this truly alters the patient's status. Surgical decompression is generally reserved for refractory cases only. Schon and Baxter[596] reported a case involving a 19-year-old college sprinter with anterior tarsal syndrome attributed to an osteophyte on the dorsum of the talus, which was surgically excised. The patient's symptoms resolved, and he has reportedly returned to training. Literature documenting the efficacy of surgical intervention otherwise appears to be lacking at this time.

Superficial Peroneal Neuropathy

Superficial peroneal entrapment neuropathy was reportedly first described by Henry in 1945 and termed "mononeuralgia in the superficial peroneal."[600,613,614] It is an infrequently diagnosed and documented condition. The average age is reported to be 36 years, but ranges from 15 to 79 years. In a subpopulation, specifically in athletes, superficial peroneal neuropathy has been diagnosed at an average age of 28 years. Men and women have been documented to experience this problem with equal incidence. Of the athletes, runners have been most frequently diagnosed with this neuropathy. It has also been documented in individuals engaging in soccer, tennis, racquetball, hockey, and dancing.[596,613–616] The most common site of compression injury is typically described as occurring at the junction of the middle and distal thirds of the leg where the superficial peroneal nerve surfaces through the crural fascia.[596,600,615]

Anatomic Considerations

The superficial peroneal nerve, a branch of the common peroneal nerve, passes through the anterolateral compartment of the leg and innervates the peroneus longus and brevis muscles. The nerve lies between the peroneus longus and the extensor digitorum muscles proximally. Its subsequent course takes it between the anterior intermuscular septum and the fascia of the lateral compartment. It then pierces the deep fascia and emerges to a subcutaneous route in the leg about 10.5 to 12.5 cm above the lateral malleolus.[596] This point is typically at the level between the middle and distal thirds of the leg. Thereafter, it divides into lateral and medial terminal cutaneous branches, also referred to as the intermediate dorsal cutaneous and the medial dorsal cutaneous nerves.[596,617] The medial terminal branch supplies the medial side of the dorsum of the foot, as well as the dorsal aspect of the medial side of the great toe and of the adjacent sides of the second and third toes. The lateral terminal division supplies sensory fibers to the lateral aspect of the dorsum of the foot and the adjacent sides of the third, fourth, and fifth toes.[617] The innervation of the toes typically extends to the level of the IP joints.[422] The cutaneous innervation of the superficial peroneal nerve also includes innervation to the skin of the anterolateral aspect of the leg.[600]

Etiology

Pecina and colleagues[600] point to trauma as the most common proposed etiology of superficial peroneal neuropathy. Styf cites literature indicating that local trauma has been reported in about 25% of patients with compression of this particular nerve.[613]

Schon and Baxter[596] note that most cases have reportedly shown that the edge of the deep fascia compresses against the superficial peroneal nerve as it pierces this latter structure. In the presence of muscle herniation through the fascia due to fascial defects, they feel the nerve is likely to experience focal compression injury.[596,611] Styf[613] points out that exercise creates sufficient pressure to herniate muscle tissue through fascial defects with resulting compression of the nerve. McAuliffe and colleagues[618] reported a case of a young woman who experienced bilateral superficial peroneal nerve entrapments as the result of fat herniation through fascial defects in the lateral compartments of both legs. In each leg, the nerve was entrapped against the fascia by a nodule of fat and was noted to have focal, fusiform swelling. McAuliffe and colleagues[618] also cite work by Banerjee and Koons[619] who documented unilateral superficial peroneal nerve entrapment exacerbated by exercise, reportedly attributable to ankle inversion injury or to the wearing of high boots.

Styf and Korner[620] note that there is a fibrotic, relatively unyielding tunnel through which the superficial peroneal nerve passes. It is located in the corner between the anterior intermuscular septum and the fascia over the lateral compartment. In the presence of edema following trauma, it is their contention that this anatomic feature predisposes an individual to a local "mini compartment syndrome." This tunnel is typically described as being of low compliance and about 1 cm in length; however, Styf[613] and Pecina et al[600] cite surgical evidence indicating it may extend from 3 to 11 cm in length.

Schon and Baxter[596] feel that the recurrent stretching of the superficial peroneal nerve that ostensibly occurs with chronic ankle sprains is a major factor in causing injury to this nerve. Pecina and colleagues[600] and Lowdon[614] also support the role of stretch injury in causing superficial peroneal nerve injury. They note that the fact that the nerve is fixed makes it vulnerable to the stretch trauma that purportedly occurs with forced, abrupt inversion and plantar flexion at the ankle. Styf and Korner[620] have documented superficial peroneal nerve entrapment as a complication of fasciotomy performed to address chronic anterior compartment syndrome. Schon and Baxter[596] presented a case of a female tennis player who had a superficial peroneal nerve entrapment most likely attributable to compression by a ganglion cyst. Styf reported one case of surgically documented superficial peroneal nerve entrapment by scar tissue following an earlier fasciotomy to decompress the latter nerve.[613]

Clinical Presentation

The typical patient presents with longstanding complaints of pain involving the lateral leg and dorsum of the foot. About one third reportedly have additional complaints of numbness and paresthesias in the latter distribution.[596] As noted previously, approximately 25% have a history of preceding trauma.[613] Ankle sprains are cited as the most common form of possible precipitating injury.[596] Although ankle eversion weakness may be associated with entrapment of this nerve, a review of the literature fails to document weakness as a presenting complaint. It is possible that the presence of chronic compression injury of the superficial peroneal nerve actually predisposes an individual to frequent ankle sprains. The sprains would then be an indirect manifestation of ankle weakness. Exercise usually exacerbates the symptoms. Nocturnal exacerbations are reportedly uncharacteristic.[596]

Patients may present with hypesthesia in the sensory distribution of the superficial peroneal nerve, but Schon and Baxter[596] report that diminished sensation to light touch and pinprick is actually atypical. Patients may also have ankle eversion weakness. A Tinel's sign may be elicited with a light tap over the site where the nerve emerges from the deep fascia, with radiation to the dorsum of the foot and toes. Point tenderness and a palpable bulge or fascial defect may also be detected at the point the nerve pierces the fascia.[596]

Styf[613] advocates the use of three provocative tests to determine if a superficial peroneal entrapment neuropathy is present. One involves applying pressure at the point the nerve emerges from the fascia (at the junction of the middle and distal thirds of the lateral leg) while the patient simultaneously actively dorsiflexes and everts the foot against resistance. The second test involves passive plantar flexion and inversion of the ankle without applying pressure at any point along the nerve course. The final and third test is performed by maintaining passive stretch in plantar flexion and inversion while the nerve is percussed along its subcutaneous route. A positive test with any one of these maneuvers is manifested by the precipitation of pain or paresthesias in the lateral leg or dorsum of the foot and ankle. Styf contends that a positive test suggests superficial peroneal entrapment neuropathy and should prompt further neurophysiologic investigation for confirmation.[613] His diagnostic criteria for diagnosis of the latter nerve entrapment include decreased sensibility and pain over the dorsal aspect of the foot at rest or during exercise, along with at least one positive test among the three aforementioned tests.[613]

Differential Diagnosis

Anteroposterior, lateral, and oblique radiographs of the leg and ankle should be performed to rule out the possibility of a stress fracture or bone tumor. An electrodiagnostic study should be performed to confirm the presence of a superficial peroneal nerve entrapment neuropathy, as well as to exclude the possibility of a more proximal peroneal nerve injury or an L-5 radiculopathy. A measure of the sensory action potentials of the cutaneous branches serving the dorsum of the foot can be performed. This technique is potentially beneficial in identifying entrapment of the superficial peroneal nerve at the point it emerges from the fascia or in isolating more distal involvement of the cutaneous branches.[422] Injection of a local anesthetic, such as 1% xylocaine, along with cortisone in the region of suspected entrapment may be both diagnostic and therapeutic.

Treatment

Avoidance of potential sources of repetitive trauma or stretch injury should be the initial goal. Running activities, in particular, may need to be curtailed or reduced. If signs and symptoms are mild and there is a perceived need to continue such activity by an athlete, then use of supportive footwear or stabilizing foot orthotics may be sufficient to address the problem. Specifically, shoes or foot orthotics providing appropriate arch supports and external heel counters may add sufficient ankle stability to minimize the potential for recurrent ankle sprains. Occasionally, an aircast brace may be warranted if only mild additional ankle mediolateral stability is necessary. If signs and symptoms persist, or if more significant impairment is noted, complete avoidance of exacerbating exercises or sport participation may be necessary. Injection of a local anesthetic with cortisone at the site of suspected entrapment may provide prompt and lasting relief. A trial of NSAIDs may offer some relief.

Extrinsic compression of the cutaneous branches of the superficial peroneal nerve is rarely documented. However, if such is suspected by history and examination, avoidance of overly tight lacing of shoes, excessively tight boots, or other poorly fitting footwear may prove beneficial.

If conservative measures fail, fasciotomy of the fascial tunnel through which the superficial peroneal nerve passes is advocated as a potentially effective means of treating this problem.[600] The aforementioned young woman presenting with bilateral superficial peroneal nerve entrapment neuropathies was successively managed with decompression of the nerve by bilateral, local fasciotomies.[618] Styf[613] studied 21 patients with documented superficial peroneal nerve entrapment. Each had been managed with fasciotomy and neurolysis. Of the 19 reviewed at a later date, 9 reported complete satisfaction, while another 6 had improvement but were unsatisfied since improvement was insufficient to allow performance of their athletic activity. Three of the 19 reported no change and one was actually worse.

Sural Entrapment Neuropathy

Sural entrapment neuropathy is infrequently diagnosed. It reportedly has no particular age or gender distribution.[596] Although entrapment may occur at any point along the course of the nerve, it most commonly occurs at the point the nerve exits the fascia in the leg 20 to 25 cm above the base of the heel.[422,600]

Anatomic Considerations

The sural nerve is a branch of the distal sciatic nerve. It courses through the posterior compartment of the leg and surfaces through the fascia. As previously noted, this occurs at 20 to 25 cm above the base of the heel.[422] It then passes down the lateral aspect of the calf and posterior to the lateral malleolus. It innervates the lateral part of the heel and sole of the foot up to the base of the fifth toe.

Etiology

Sural nerve injury may occur as the result of laceration trauma or compression injury. It reportedly occurs most frequently in runners.[596] The nerve is particularly vulnerable to compression at the point it pierces the fascia. This is a common site for partial or complete excision biopsy of the nerve.[422] Sural nerve compression has also been reported in athletes with fractures of the base of the fifth metatarsal incurred with severe ankle plantar flexion and inversion injuries.[600,621]

Recurrent ankle sprains with accompanying scar formation have been postulated to cause sural entrapment neuropathy.[600] Schon and Baxter[596] report a case of a male middle-distance runner with a two-year history of pain along the Achilles tendon. In the latter case, the sural nerve was found to be tightly adherent to an aponeurotic band behind the Achilles tendon. The patient responded successfully to surgical release of the aponeurosis and lateral displacement of the sural nerve.

Another potential cause of sural nerve entrapment includes ganglion of the peroneal sheath or calcaneal cuboid joint.[596,622] Schon and Baxter[596] also cite a case report by Husson and colleagues in which an individual was found to have entrapment of the sural nerve by myositis ossificans circumscripta at the musculotendinous junction of the Achilles.

Clinical Presentation

Patients typically complain of progressive increase in symptoms of paresthesias and shooting pain down the lateral aspect of the leg with radiation to the lateral aspect of the ankle and foot. They may have a history of recurrent ankle sprains. As previously noted, they may present with a history of running for exercise or sport. An increase in mileage or intensity of running may be temporally related to onset of their symptoms.

There may be decreased sensation to light touch or pinprick in the distribution of the sural nerve, but it is typically limited to an area a few centimeters below the lateral malleolus.[422] A Tinel's sign may be elicited with percussion at the site of entrapment. This sign most commonly occurs at the nerve's exit from its fascia. It is characteristically manifested by radiating paresthesias beyond the area of sensory loss to the lateral aspect of the foot.[422] After nerve biopsy, permanent sensory loss or persistent painful paresthesias are potential complications.

Differential Diagnosis

Electrodiagnostic studies, specifically a sural sensory NCS, may be helpful in confirming the presence of the entrapment neuropathy and in excluding the possibility of a sciatic nerve lesion, an S-1 radiculopathy, or a peripheral neuropathy. Radiographic evaluation of the leg, ankle, or foot may be warranted if pain is the sole or primary complaint. The latter studies would serve to exclude the possibility of a stress fracture or bone tumor.

Treatment

Cessation or modification of a suspected precipitating activity, such as running, should be recommended. The use of orthotics or bracing has not been advocated in the literature, presumably because it has limited application in this form of entrapment neuropathy. A trial of NSAIDs is warranted. If unresponsive to conservative management, surgical exploration and decompression at the site of entrapment merits consideration.

Digital Neuropathies

Digital neuropathies secondary to interdigital neuromas are relatively common, particularly in the athletic population. Runners and dancers appear to be the most susceptible to this problem.[596,600] The elderly population and women appear to manifest this form of neuropathy with greater frequency than others. In the former, it may simply reflect the effects of chronic repetitive nerve irritation that may come with increasing age, and in women presumably due to wearing of high heels and tight, pointed shoes.[596]

Morton's toe neuroma is a specifically described entrapment of an interdigital nerve. This interdigital neuroma is typically described as occurring between the third and fourth digits of the foot, although it may also occur in the second web space.[422,596]

Anatomic Considerations

The medial plantar nerve is the larger of the two terminal branches of the tibial nerve. In its distal segment, it divides into four plantar digital nerves. The plantar digital nerves are distributed to the plantar aspect of the medial three and one-half toes. In addition, they also innervate the distal aspect of the extensor surface of these toes. The first plantar digital nerve innervates the medial surface of the plantar aspect of the great toe, while the other three provide nerve supply to adjacent sides of the great, second, third, and fourth toes, respectively. The second plantar nerve also supplies the first lumbrical muscle.[617]

The lateral plantar nerve divides into a superficial and a deep branch in its distal segment. The superficial branch then divides into two plantar digital nerves. One of the latter nerves supplies the lateral side of the little toe, the flexor digiti minimi muscle, and the two interosseus muscles of the fourth space. The other digital nerve innervates the adjacent sides of the fourth and fifth toes, as well as the distal part of the extensor surface of the corresponding distal phalanges. The deep branch of the lateral plantar supplies the interossei of the first three spaces, the two or three lateral lumbricals, and the adductor hallucis muscle.[617]

Etiology

Morton is noted to have described the condition of Morton's toe neuroma in 1876 and felt that it was caused by pinching of the lateral plantar nerve. However, it took until the 1940s before it was recognized that the signs and symptoms were actually due to a neuroma of the interdigital nerve secondary to entrapment.[422] Review of the literature by Guiloff and colleagues[623] indicate that an early investigator felt that this nerve lesion was due to ischemic insult, noting that a number of investigators since that earlier time support the contention that it is, in fact, an entrapment neuropathy.

The sharp anterior edge of the deep plantar fascia forms the intermetatarsal ligament and it is this structure that is felt to cause focal injury to a digital nerve.[422] As previously noted, the toes are forced into dorsiflexion at the metatarsophalangeal joint when high heels and tight, pointed shoes are worn. This posture is felt to force the nerve against the intermetatarsal ligament with resulting compression neuropathy. The repetitive, extreme dorsiflexion that occurs at the metatarsophalangeal joints of dancers and runners presumably causes repetitive microtrauma to the plantar nerve when it is repeatedly forced up against the distal edge of the intermetatarsal ligament.[596] Repetitive or sustained squatting is also associated with excessive dorsiflexion at the metatarsophalangeal joints and thus, is considered a possible cause of plantar nerve entrapment neuropathy.[422]

It has also been suggested that runners with a hypermobile first metatarsal are more susceptible to developing calluses under the second and third metatarsal heads. A neuroma may develop as a consequence of the pressure created by the calluses.[596] Schon and Baxter[596] feel excessive pronation may result in increased dorsiflexion of the third metatarsal relative to the fourth. As a consequence, the nerve is felt to be more vulnerable to impingement in the presence of hyperpronation.

Clinical Presentation

Burning pain is characteristic of digital neuropathies due to interdigital neuromas. The pain is typically in the plantar region and exacerbated with prolonged standing or running, particularly sprinting or long-distance running.[422,596] Some relief is typically obtained with rest, gentle foot massage, and elevation of the affected lower extremity. Aching, cramping, or sharp pain with radiation to the toes has also been recorded.[596] Others have reported intermittent pain described as a stabbing, shooting, or pricking discomfort "like needles." Still others complain of a piercing, hot pain.[623]

Point tenderness with palpation over the metatarsal head or more typically with pressure in the space between the metatarsal heads, usually between the third and fourth toes, is a reproducible finding. Less frequently, the tenderness may be elicited with pressure between the second and third toes.[422,596] Focal swelling may be detected at the site of tenderness. Exquisite pain may be elicited by squeezing the metatarsal heads together.[596] Other provocative tests to reproduce symptoms also include passive dorsiflexion of the toes along with firm palpation of the intermetatarsal space and squatting by the patient. There may be a hypermobile first metatarsal and calluses under the second and third metatarsal head, conditions that have been suggested to predispose individuals to developing interdigital neuromas. Although not consistently present, impaired sensation in the medial half of the fourth toe may be present; however, other objective neurologic deficits are reportedly not typically found.[422]

Differential Diagnosis

Stress fractures, arthritic disease, and bone tumors should be excluded with appropriate radiographs. Metatarsal capsulitis due to strain, contusion, or a subluxed metatarsophalangeal joint, as well as adventitious bursas, sesamoiditis, plantar fasciitis, and peripheral neuropathy may mimic some of the signs and symptoms found with interdigital neuromas; however, an appropriate history and careful physical examination will generally exclude these conditions.

Electrodiagnosis

An electrophysiologic technique for assessing the integrity of the interdigital nerves has been developed by Oh and colleagues.[624] It includes electrical stimulation of the great toe and fifth toe with ring electrodes. The recording electrode is provided by placement of a near-nerve needle at the ankle. This technique permits simultaneous stimulation of two branches of the interdigital nerves.[422,624] Dawson and colleagues[422] point out that decrease in the amplitude of the sensory action potential is characteristic of an affected interdigital nerve. Guiloff applied a similar electro-physiologic technique to assess patients presenting with Morton's metatarsalgia. They concluded that this form of assessment with the technique they employed was not advisable for routine testing of such patients nor a replacement for a sound clinical examinaton.[623] Katz[625] acknowledges that such electrophysiologic studies are available, but notes that success in obtaining consistent results is variable.

Treatment

Conservative treatment for interdigital neuromas initially includes avoidance of the physical activity presumed to precipitate or exacerbate the symptoms. It also includes prescription of an NSAID, local massage, as well as shoe modifications and the use of foot orthotics. Typically, metatarsal pads and shoes with an ample toe-box are prescribed. At times, extra-depth shoes are employed. Use of high-heels and pointed shoes are discouraged. A trial injection of cortisone with local anesthetic occasionally offers lasting relief. However, Lillich and Baxter[626] point out that use of metatarsal pads and local anesthetic and cortisone injections are successful in relieving symptoms less than 50% of the time.

If a patient's symptoms are refractory to the aforementioned conservative measures, then excision of the neuroma in toto is advocated. Dawson and colleagues[422] report that this particular surgical intervention has a high success rate with the primary residual deficit limited to the sensory distribution of the excised nerve. As previously noted however, the sharp anterior edge of the deep plantar fascia forms the intermetatarsal ligament and it is this structure that is felt to cause focal injury to a digital nerve.[422] This constitutes the rationale for releasing the latter ligament without excision of the neuroma as advocated and practiced by Gauthier. He reportedly obtained successful results in over 200 patients managed in this fashion.[422,627]

CONCLUSION

Traumatic injuries to the peripheral nerves pose complex challenges to the military physician. While the etiology and specific pathology may be wide ranging, it is not uncommon for even mild injuries to be severely disabling, if not properly diagnosed and treated. Comprehensive management requires an extremely detailed assessment, and may require both electrodiagnostic testing and surgical exploration.

Treatment of nerve injuries must be multidisciplinary and must consider all aspects of the inherent disability. Pain control is of primary importance. Little else in terms of functional restoration will be accomplished until pain is brought down to tolerable levels. As neural regeneration may take months to become clinically evident, even after surgical intervention, protection of the affected area from complications of disuse and immobility are essential. Rehabilitation efforts, including the enhancement of strength, flexibility, sensory discrimination, and dexterity, must commence early in the treatment course to harness and improve residual function and to set the stage for more complete and successful recovery as reinnervation occurs.

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Chapter 10

REHABILITATION MANAGEMENT OF BURN CASUALTIES

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INTRODUCTION

CLASSIFICATION
  Thermal Injuries
  Chemical Burns
  Electrical Injuries

FACTORS IN BURN SEVERITY
  Age
  Burn Size
  Burn Depth
  Associated Illnesses and Injuries

TREATMENT OF BURNS
  Topical Treatment
  Complications
  Medical/Surgical Considerations
  Emergency Burn Care
  Outpatient Clinic vs Specialized Inpatient Treatment
  Early Life-Support Evaluation and Management
  Fluid Resuscitation for Burn Shock
  The Nonhealing Burn Wound
  Burn-Wound Excision
  Tangential Excision
  Excision to Fascia
  Enzymatic Debridement
  Donor Site Selection
  Skin Grafts
  Biological Dressings
  Heterograft and Synthetic Dressings
  Donor Sites
  Goals of Rehabilitation Management

MANAGEMENT OF BURN CASUALTIES
  Acute Phase of Healing
  Immobilization Phase of Wound Care
  Wound Maturation Phase of Healing
  Nutritional Care of Burn Patients
  Neurologic Problems in Burn Patients
  Plastic Surgery Strategies and Rehabilitative Considerations

CONCLUSION

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INTRODUCTION

Most burn casualties require evacuation from the theater of operations. Ideally, severely burned casualties will be evacuated rapidly to burn centers, such as the U.S. Army's Institute of Surgical Research at Fort Sam Houston, Texas. Actual battlefield conditions, including dehydration, decreased nutritional balance, fear, other psychological stressors, and extremes of climate may contribute to the severity of the burn wounds and also hinder the healing process. Fluid for wound cleansing, topical antimicrobials, effective analgesics, and bandages are not easily carried in a field pack. The mobile army surgical hospital is not appropriate to care for burn wounds, which heal in two to three weeks or which need grafting and rehabilitation.

This chapter will primarily address rehabilitation care of the casualty who is transferred back to a burn center in the continental United States. After a major burn injury, the casualty benefits from a comprehensive rehabilitation treatment plan to quickly recover the maximal possible preburn status of strength, endurance, active range-of-motion (AROM), coordination, and mental functioning. The rehabilitation plan will assist military personnel to achieve independent self-care and return to active duty as soon as the wounds are healed and durable. For optimal final outcome, the constant goal of the casualty, family, surgeon, and therapists must be achieving the soldier's preburn level of active function and return to duty, despite the burn injury.

The following presentation is divided into four sections, the first three introductory sections discuss (1) classification, (2) pathophysiology, and (3) medical surgical considerations of burns. The fourth section deals with the management and rehabilitation of burn casualties; and is divided into (*a*) the acute phase, (*b*) the immobilization phase, and (*c*) the maturation phase of wound healing.

CLASSIFICATION

Burns are classified by mechanism of causation: thermal, chemical, electrical, and radiation; the depth of injury; the location of injury; and associated other injuries.

Thermal Injuries

Heat injury accounts for 85% to 90% of civilian burn victims in the United States, with chemical and electrical making up the majority of the remainder. Flame burns are the most common casualties seen at a time of conflict and may be caused by petroleum products including napalm, jet fuel, or gasoline. Contact with hot liquids results in scald burns; grease burns are common among cooks, kitchen workers, and mechanics. Superheated steam burns with inhalation complications occur from ship or submarine boilers.

Cold injury has some of the characteristics of thermal injury and most commonly occurs when a distal part is exposed to low environmental temperatures. Volatile liquids can produce localized freezing injury to tissues, especially if the liquids are allowed to escape and change rapidly to a gaseous state. The severity of injury depends on the intensity and duration of exposure and the care taken to avoid damaging the cell nucleus while it is in crystal form. The depth of injury can rarely be determined before rewarming. Fortunately, the degree of gangrene is often less than initially feared. For this reason, unlike heat injuries, amputation is delayed until the extent of gangrene is certain. Unlike heat injury, after healing there is frequent permanent increase in vasoconstrictor tone resulting in hyperhidrosis and abnormal sensitivity to cold. Pain and paresthesia are common from residual neuritis. Long term joint chondral changes are common, especially in the small finger joints.

Chemical Burns

Most chemical burns are caused by acids, alkalis, or vesicants. Except for white phosphorus, most chemical burns should be immediately treated with copious water lavage for a minimum of 30 minutes; it is inappropriate to waste valuable time searching for a specific neutralizing agent. Neutralization generates heat and increases the depth of burn. Powdered chemicals should be brushed away prior to water lavage to decrease the quantity of exposure. Lavage is continued until skin pH has returned to neutral and pain has abated. Lavage of the eye is performed with an irrigating lens.

Chemical agents produce direct tissue damage by a variety of reactions. Acid burns tend to be superficial in depth whereas alkali burns tend to burrow into the tissues and cause more significant destruction. Acids cause coagulation necrosis (Fig-

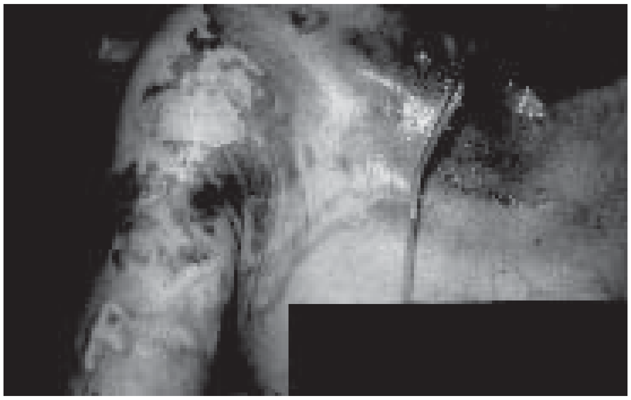


Fig. 10-1. Coagulation necrosis of shoulder, secondary to acid burn.

ure 10-1). Alkalis cause liquefaction necrosis. Once the initial care, neutralization, and removal of the offending agent are completed, the rehabilitative care is much the same as that for a thermal burn.

White phosphorus, which ignites on contact with air, is covered with water or saline until the wound can be debrided of all phosphorus. Hydrofluoric acid requires copious water lavage followed by topical calcium gluconate. Calcium gluconate injected into the area may decrease the severity of the injury. The use of intraarterial calcium gluconate for wounds of the hands and feet is beneficial.

Mustard is the vesicant most likely to be used in warfare. It is rapidly absorbed by the skin, conjunctiva, and mucous membranes, and within minutes, irreversibly combines with tissue proteins. Ophthalmic injuries are best treated with copious water irrigation. Skin is decontaminated with 0.5% hypochlorite. In general, the skin burns are superficial and heal without difficulties. Injuries to the mucosal linings are much more serious and disable the soldier for combat.

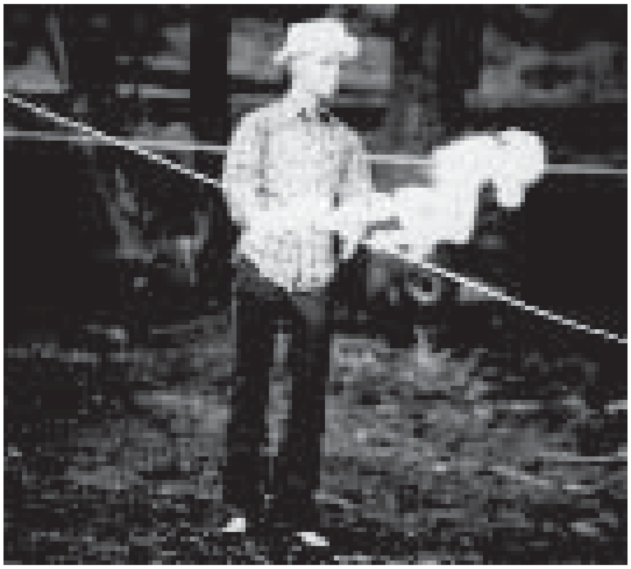


Fig. 10-2. Demonstration of electrical arc ("Jacob's Ladder" effect).

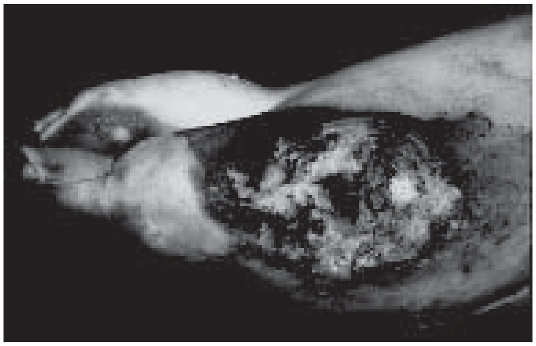


Fig. 10-3. Electrical exit injury.

Electrical Injuries

Electrical injuries result from the conduction of electrical energy through tissue or from heat which is released as the current arcs through the air (Figure 10-2). Current arcs may generate temperatures as high as 3,000°C; as they arc, they ignite clothing, which results in a combination electrical (Figure 10-3) and thermal injury (Figures 10-4 and 10-5).

Injuries caused by low voltage current (< 1,000 V) are occasionally fatal due to immediate ventricular fibrillation. Survivors rarely have significant tissue damage.

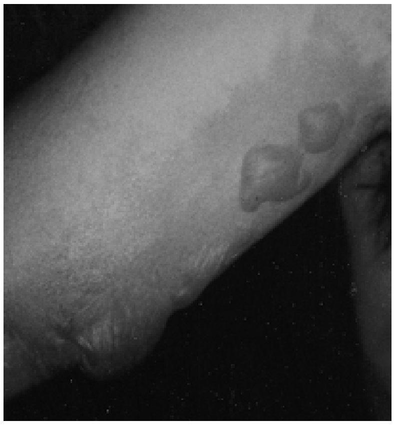


Fig. 10- 4. Superficial partial thickness burn blisters from a thermal burn.

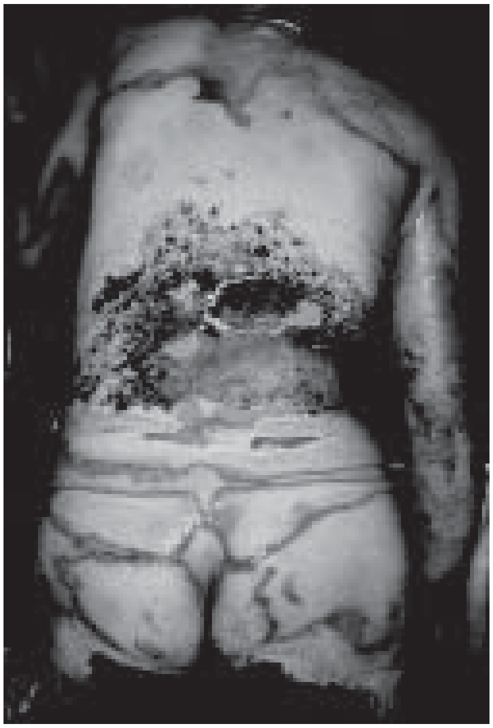


Fig. 10-5. Mixed deep partial and full thickness thermal burn.

High voltage (> 1,000 V) current can damage tissue anywhere along its route. Electrical injuries are heat related. Electrical energy is converted to heat energy as expressed by Joule's law: power or heat equals amperage squared times resistance ($P=I^2 \bullet R$). The higher the voltage and/or the amperage, the greater the amount of heat that will be generated and the more serious will be the problem. Measurement of tissue temperature experimentally reveals that tissue temperature is highest directly underneath and adjacent to the contact site of the entrance and exit wounds. Deep tissue destruction is always greatest in areas of the body with small volume such as the fingers, toes, wrists, or ankles. The farther away the tissue is from the contact point, the less the current density is, and, therefore, the less heat is generated. Various tissues have different resistance to current flow with nerve and blood vessel having low resistance and cartilage and bone having high resistance. Because bone has a high resistance, current tends to flow at its surface and, therefore, temperature is greatest at the periosteum. Thus, muscle damage is often extensive adjacent to the bone, and many times the periosteum and portions of the outer cortex of the bone may not be viable. For these reasons, an electrical injury may not appear severe initially since only the entrance and exit (see Figure 10-3) sites show visible damage, yet the casualty may have severe limb damage that will necessitate limb amputation.[1]

A sensitive indicator of total muscle damage following electrical injuries is the serum creatine kinase (CK). In a recent study, patients with a total CK of under 400 international units (IU) had no significant tissue loss; a few patients with total CK concentration ranging between 400 IU and 2,500 IU had digit amputations or skin grafts. Patients with a total CK greater than 2,500 IU had a high risk of major amputation; and those with a CK total greater than 10,000 IU had an 84% risk of major amputation or permanent neurologic deficit.[2]

FACTORS IN BURN SEVERITY

Age

The severity of burn injury is determined by patient age, total body surface area (TBSA), associated injuries, and to a lesser extent, burn depth and associated illnesses.[3] The very young and the very old do not tolerate illness and trauma, particularly burn trauma, as well as those in the prime of their lives. In the case of burns, this "prime" lies somewhere between 10 and 50 years of age. Persons at the extremes of age generally are more fragile physiologically and tolerate poorly the massive fluid shifts and infectious complications associated with the burn and its treatment. The American Burn Association, in its 1992 Postgraduate Course recognized this effect of aging and recommended that patients with smaller burns, if young or old, be treated in a burn center. During combat, available resources must be expended on those individuals with the greatest chance for survival.

Military burn casualties typically range from 18 to 40 years of age. In contrast, approximately 50% of patients admitted to a civilian regional burn center will be under 18 or over 50 years of age.

Burn Size

Evaluation and treatment of the burn requires an accurate assessment of burn size. The three most commonly used methods of determining burn size are the Rule of the Palm, the Rule of 9s, and the Lund and Browder chart. The Rule of the Palm states that the patient's palm, excluding the fingers and the thumb, is approximately equal to 0.5% of

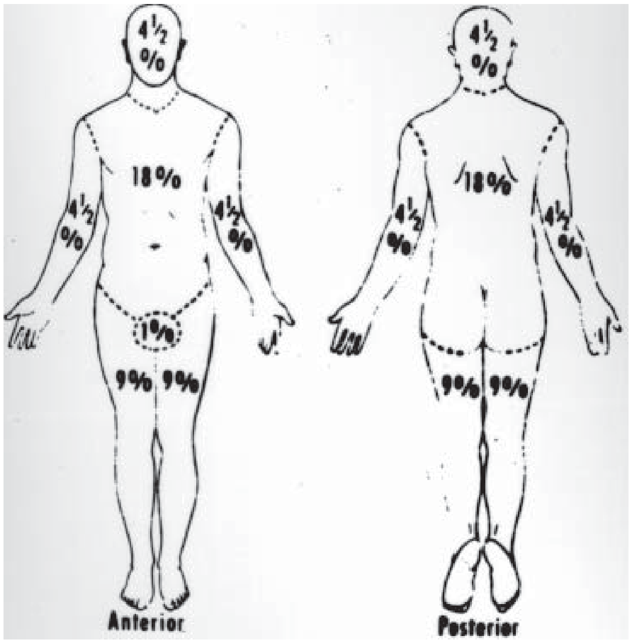
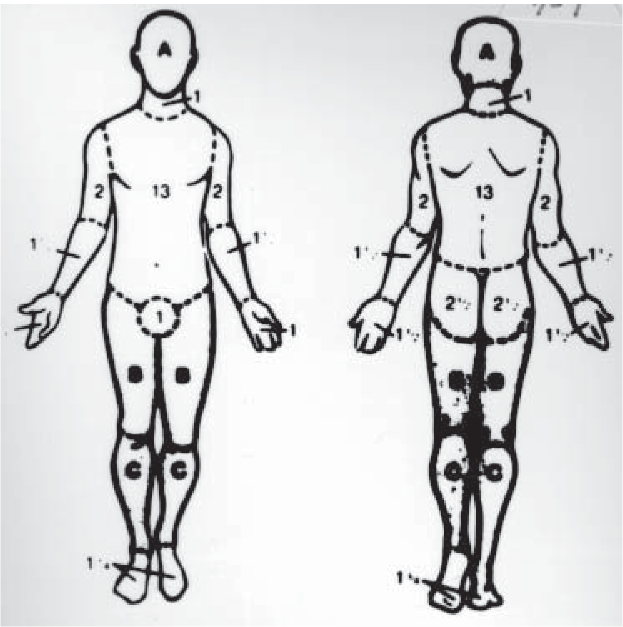


Fig. 10-6. The Rule of 9s. Adapted from Bowen TE, Bellamy RF, eds. *The Emergency War Surgery NATO Handbook*. 2nd rev. Washington, DC: Department of Defense; 1988.



Relative Percentages of Areas Affected by Growth

Age in Years	Half of Head (A)	Half of One Thigh (B)	Half of One Leg (C)
Infant	9½	2¾	2½
1	8½	3¼	2½
5	6½	4	2¾
10	5½	4¼	3
15	4½	4½	3¼
Adult	3½	4¾	3½

Fig. 10- 7. The Lund and Browder chart. Adapted from Cioffi WG, Jr, Rue LW III, Buescher TM, Pruitt BA. A brief history and the pathophysiology of burns. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries*. Part 1, Vol 5. In: *Textbook of Military Medicine*. Washington, DC: Office of The Surgeon General, Department of the Army, and Borden Institute; 1991: 341.

the patient's TBSA. The Rule of the Palm may be used as a quick estimate of burn surface area involvement and can be easily applied in the emergency room or field setting for determination of small burns or burns of scattered areas.[4]

The Rule of 9s (Figure 10-6) is a convenient way of estimating adult total body surface area. Developed in the 1940s by Pulaski and Tennison, it is easily remembered because it divides the body surface into areas of 9% or multiples of 9%. The head and neck equal 9%, each arm and hand is 9%; the anterior and posterior trunk are two 9s, or 18% each; each leg is two 9s, or 18%; and the perineum is 1%. The Rule of 9s is easily applied in field situations, but we believe it to be relatively inaccurate.[5]

The Lund and Browder chart (Figure 10-7) was developed nearly 50 years ago and more accurately defines burn size, and is used in most burn centers. This chart assigns a percent of surface area to body segments.[6] It takes into account the disproportionate growth of trunk, head, and lower extremities in children. However, the chart is not always readily available and is too complex to commit to memory.

Burn Depth

Burns historically have been classified into degree categories: first, second, third, and fourth (Figure 10-8). First and second degree burns are partial thickness injuries, whereas third degree burns are full thickness. First degree burns involve only the epidermis, are erythematous or deeply tanned in appearance, and do not blister. Sunburn (Figure 10-9) is a typical first degree burn, heals in 3 to 6 days, and has no long term sequelae.

A second degree burn involves both the epidermis and dermis. A superficial second degree (superficial partial thickness) burn (see Figure 10-4) blisters, is moist and erythematous, and reepithelializes in less than 3 weeks in adults or less than 2 weeks in children. Superficial second degree burns are best treated with daily dressing changes and allowed to heal without surgical intervention. A

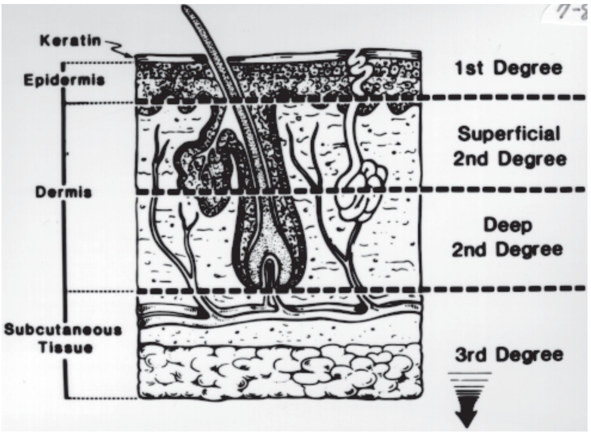


Fig. 10-8. Skin diagram illustrating depth of injury.

superficial second degree burn may heal with minor color and texture changes, but it does not develop hypertrophic scarring.

In contrast, a deep second degree (deep partial thickness) burn (see Figure 10-5) is cream colored or white beneath the blisters, takes longer than 3 weeks to heal in adults (> 2 weeks in children under 10 years of age), and is best treated with early excision and grafting. A spontaneously healed deep second degree burn will have fragile skin and may develop severe scarring.

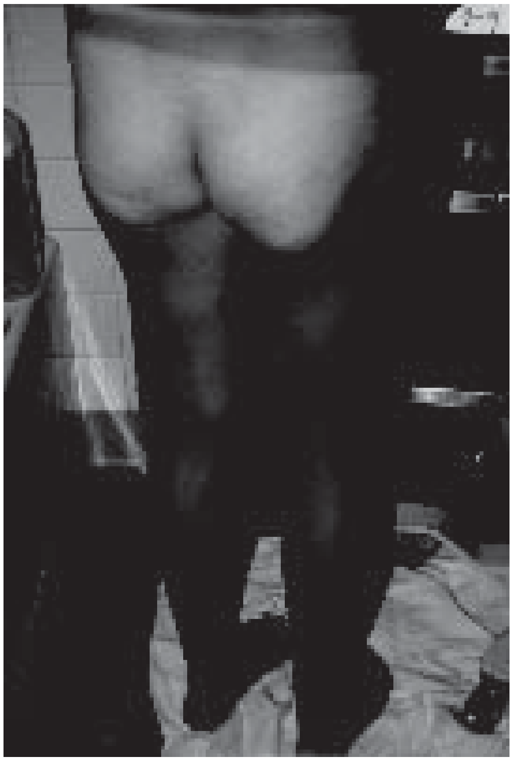


Fig. 10-9. Sunburn injury.

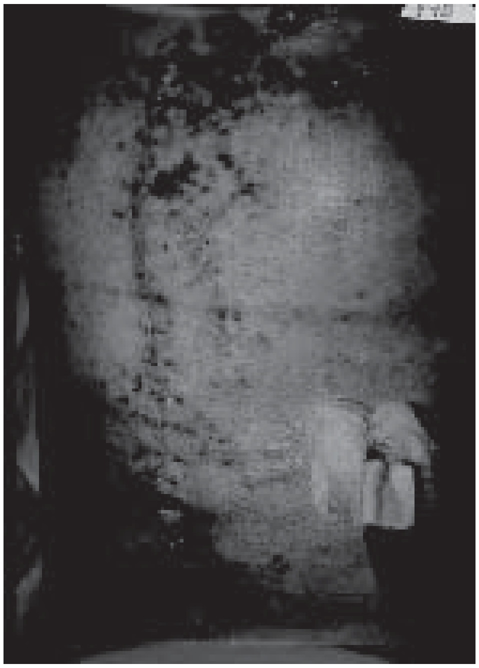


Fig. 10-10. Burns secondary to explosion and gunshot wound. Note that the explosive device caused fragment wounds and burns. Intraabdominal pathology necessitated splenectomy and repair of gastric lacerations.

A third degree (see Figure 10-5) (full thickness) burn involves the entire depth of the skin, destroys both the epidermis and dermis, and must heal from the margins. A third degree burn larger than 3 cm in diameter is best treated with early excision and grafting.

A fourth degree burn occurs when damage is deep to the skin and involves muscle, bone, and other deeper tissues.

Associated Illnesses and Injuries

Most military casualties occur in healthy adults. If civilians are injured it must be remembered that chronic illnesses such as diabetes, renal failure, and chronic liver disease increase their mortality. A psychiatric illness or chemical dependency will complicate nursing care, interfere with surgical treatment, and impede normal rehabilitation.

Combat burn victims have a high incidence of associated injuries[7] (Figure 10-10). The acute injuries, whether due to penetrating or blunt trauma, are given first priority and will be handled in the field hospital. The patient's burn fluid resuscitation is performed during the treatment of the other injuries. Inhalation injuries complicate about 20% of burn admissions.

TREATMENT OF BURNS

Topical Treatment

Burn wounds are cleansed once or twice daily followed by application of a topical agent. For small wounds (less than 10% TBSA), a soothing ointment such as Bacitracin (Barre-National, Inc.) is a relatively inexpensive topical agent, but does not penetrate well and is ineffective against gram negative bacteria. Superficial wounds may also be treated with synthetic, semisynthetic, or biologic dressings such as Biobrane (Winthrop Consumer Products) or pigskin (Figure 10-11). When these products adhere, they provide a comfortable wound cover that does not separate until the wound has reepithelialized.

For deeper wounds, daily cleansing followed by application of silver sulfadiazine is the most commonly used treatment in the United States. Silver sulfadiazine does not penetrate well but has a good spectrum of coverage. It is easy to apply buttered over the wound or on longitudinal strips of fine mesh gauze and is the least painful agent, but sensitivity and leukopenia are not uncommon. Mafenide acetate (Sulfamyalon [Dow Hickman Pharmaceuticals]), the first wide-spectrum burn cream used, has excellent wound penetration and a broad spectrum of coverage. However, it causes marked pain and has a side effect of causing metabolic acidosis, making it a less popular agent. Most wounds are acutely treated with topical agents and daily dressing changes; wounds that will require longer than 3 weeks to heal are usually excised and grafted beginning 1 to 5 days postburn. Silver sulfadiazine is the most commonly used topical antimicrobial. Furacin (Roberts Pharmaceutical Corp.), 0.5% silver nitrate solution, povidone-iodine, and Bacitracin are also available for topical use.

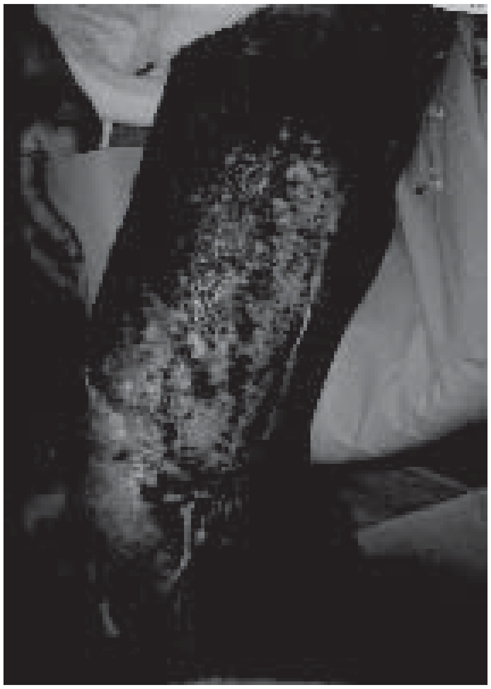


Fig. 10-11. Superficial burn healing under pigskin. Note the pigskin on posterior thigh.

Complications

Inadequate fluid resuscitation in the presence of circulating nephrotoxins (such as myoglobin) may cause renal failure. Similarly, pulmonary edema associated with excessive fluid administration occurs if the patient is not adequately monitored. Gastrointestinal disorders, including ileus, are avoided by nasogastric tube decompression of the stomach accompanied by enteral feeding through a tube placed beyond the ligament of Treitz. Curling's ulcer with erosion of the stomach and duodenal mucosa has been largely eliminated by the use of histamine-2 blockers, early enteral feeding, and antacids. Sepsis remains a common complication caused by bacterial colonization of the burn wound and the patient's immunosuppressed condition,[7] with the lungs being the most common site of fatal sepsis.[8] These complications must be understood by rehabilitative personnel as the care plan will need to be modified as the complications arise.

Medical Surgical Considerations

When the skin, the largest organ of the body, sustains significant injury, a cascade of metabolic and physiologic consequences occurs that can affect survival adversely. While providing both a fluid barrier and a thermoregulatory mechanism, the primary function of the skin is to maintain homeostasis. Intact, waterproof skin is the first line of defense against the hostile microbial world. The soldier's self-image is dependent on appearance, which has been determined by skin surface. Skin plays a vital part in bodily function. However, its importance is often underestimated, since it readily heals from minor superficial injuries with little pain or inconvenience.

Emergency Burn Care

The initial rescue and treatment of the burn victim, military or civilian, is seldom rendered by a physician but by a bystander, medic, paramedic, or other soldier. This treatment should follow the basics of first aid: (1) stop the burning process, (2) evaluate and secure the airway, and (3) treat other life threatening injuries. Chemical burns should be copiously irrigated with water or saline. If the airway is compromised (see Exhibit 10-1), the ventilation should be maintained by mask and oxygen and intubation if possible. When facial or neck edema is anticipated from fluid administration, intubation is completed prior to transfer to an aid station. The burn wound can be covered with a clean sheet or simple dressing to minimize further contamination and to help conserve body heat. While stopping the burning process, wetting and cooling the burn can rapidly result in hypothermia, a condition to be avoided. The basics of trauma emergency care apply to initial burn treatment as well as to any other injuries.

EXHIBIT 10-1

SIGNS OF AIRWAY AND PULMONARY COMPROMISE

- Facial burns with perinasal, perioricular involvement
- Singed nasal hairs, swollen lips, and tongue
- Carbon particles in mouth and sputum
- Hoarse, raspy voice
- Heavy productive cough with carbonaceous sputum
- Dyspnea, air hunger, or gasping
- Narrowing of airway on lateral cervical radiograph
- Signs of acute pulmonary edema and adult respiratory distress syndrome on chest radiograph
- Information that burn occurred in a closed space

Outpatient Clinic vs Specialized Inpatient Treatment

Recent efforts at health care cost containment have resulted in the treatment of larger uncomplicated burns in the outpatient setting. Although most military burn casualties are managed on an inpatient basis, outpatient care is possible in a garrison setting for certain soldiers with small burns. Exceptions to this trend remain in the extremes of age, an unreliable patient, or lack of family or nursing resources. The success of outpatient treatment involves good wound care by an experienced burn nurse and, later, by the fully instructed patient, family member, or buddy. Most failures of outpatient burn care do not involve wound problems, but rather are related to inadequate pain control and anxiety. Wound elevation, occlusive dressings, and liberal use of appropriate narcotics are the best means of controlling pain. The adequacy of pain control is a decision made by the patient, not the medical personnel.

There are no set criteria to determine which patients can be managed as outpatients because so many factors influence the severity of a burn. A young, otherwise healthy, individual with a 30% partial thickness burn and a good support system could not be treated at the battle front. However, he might do well as a hospital outpatient, whereas patients who are elderly, chronically ill, or alcohol or drug abusers with a 5% TBSA burn may be at risk for dying unless treated in the hospital. Cases of suspected abuse by burning, regardless of age, require admission.

Frequently used guidelines for inpatient burn treatment include:

- Greater than 10% full thickness burn.
- Greater than 20% partial thickness burn.
- Burns involving vital areas, eg, hands, feet, face, or perineum.
- Most infants and elderly—the extremes of age.
- Children with more than a 5% burn who are neglected or abused or lack adequate pain control.
- Inhalation injury.
- Most high voltage electrical injuries.
- Most chemical injuries, with special emphasis on alkali, dichromate, and fluoride burns.

The American Burn Association has identified the following[9] injuries as those requiring a referral to a burn center (Table 10-1). Patients with these burns should be treated in a specialized burn facility after initial assessment and treatment at an emergency department:

TABLE 10-1

ADMISSION CRITERIA

Age (y)	Burn Size (% TBSA)	Admission Yes	Admission No
< 10 or > 50	< 10		x
10–50	< 20		x
< 10 or > 50	> 10	x	
10–50	> 20	x	

Additional Factors Mandating Admission

- Burns of the face, hands, feet, perineum, or genitalia
- Inhalation injury
- Associated major trauma
- Major concurrent medical illness
- Electrical or chemical injury

TBSA: Total body surface area

- Second and third degree burns greater than 10% TBSA in patients under 10 or over 50 years of age.
- Second and third degree burns greater than 20% TBSA in other age groups.
- Second and third degree burns with serious threat of functional or cosmetic impairment that involve face, hands, feet, genitalia, perineum, and major joints.
- Third degree burns greater than 5% TBSA in any age group.
- Electrical burns, including lightning injury (greater magnitude of complications).
- Chemical burns with serious threat of functional or cosmetic impairment.
- Inhalation injury with burn injury.
- Circumferential burns of the extremities and chest.
- Burn injury in patients with *preexisting medical disorders,* which could complicate management, prolong recovery, or affect mortality.
- Hospitals without qualified personnel or equipment for the care of children should transfer burned children to a burn center with these capabilities.
- Any burn patient with *concomitant trauma* (eg, fractures) in which the burn injury poses the greatest risk of morbidity or mortality. However, if the trauma poses the greater immediate risk, the patient may be treated in a trauma center initially until stable, and then transferred to a burn center. Physician judgment will be necessary in such situations, and should be in concert with the regional medical control plan and triage protocols.

Early Life-Support Evaluation and Management

Burns, like any other major trauma, require that attention be directed toward the injury that is most life threatening. The burn injury associated with other major trauma may complicate diagnostic evaluation and significantly impact survival. A thorough, rapid assessment of the casualty helps direct care during the initial critical phase. This would include a history; a physical examination; and visual examination of all areas, undressed; appropriate radiography; and laboratory data.

A burn patient in acute distress on initial presentation is usually experiencing airway compromise, pulmonary insufficiency, or both (see Exhibit 10-1). Injury to the airway and lungs is more the result of inhaled toxic products of combustion rather than heat. However, someone injured in the presence of superheated steam in an enclosed space, such as a boiler room on a ship, may have burns of the airway. Inflammation and edema can rapidly obstruct the upper airway, especially in children. Early intubation may often be the prudent choice of management to avoid traumatic intubation through an edematous airway and associated complications.

Pulmonary edema may be the direct result of inhalation injury, independent of too vigorous a fluid resuscitation course. Inhalation injury can manifest itself early through impaired gas exchange with falling PO_2 and oxygen saturations, followed later by rising PCO_2. These changes can precede changes on chest radiography, sometimes by 12 to 24 hours (Figure 10-12). Inability to adequately exchange gases is an indication for intubation and controlled ventilation. Nasal intubation is generally preferred over tracheostomy for early respiratory distress.

Vascular access can be established peripherally for immediate use or centrally for more long term use. Not without its own risks, central line lumen access can more reliably deliver high volumes of fluid and medications, as well as provide a means of hemodynamic monitoring (central venous pressure) and withdrawing blood. Although an unburned site is preferable, a properly maintained line through burn tissue should still have an acceptably low infection rate. A Swan-Ganz line can provide supplemental data to guide the complicated resuscitation, such as in an elderly individual with coronary artery disease or congestive heart failure. In-

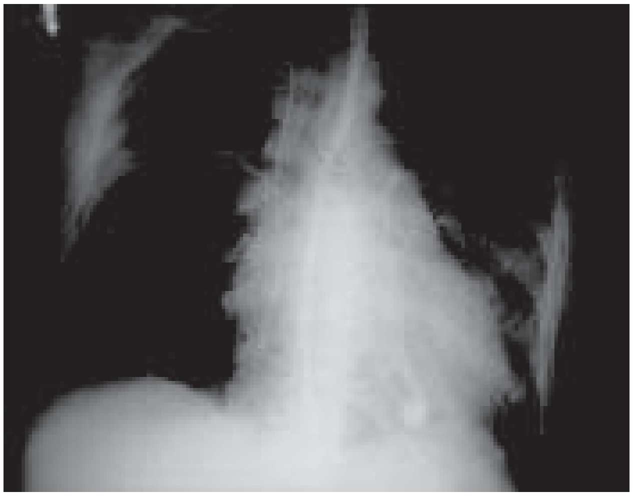


Fig. 10-12. Early chest radiograph showing changes associated with smoke inhalation.

vasive (Swan-Ganz) monitoring may be necessary to optimize resuscitation of the seriously burned patients because there is no correlation between fluid challenges and changes in vital signs and urine output.[10] Invasive monitoring carries an increased risk of sepsis, especially in burn causalities, and therefore, it must be used with caution and for a limited time.[11] The patient with cardiovascular instability or inhalation injury often benefits from an arterial line where the data provided and the blood withdrawing capabilities eliminate repeated arterial punctures.

An indwelling urinary catheter is needed for any major burn resuscitation as urinary output, a good indicator of (renal) tissue perfusion, is generally accepted as a reflection of adequacy of resuscitation. Adequate pediatric urinary output is between 0.5 and 1.0 cm^3/kg/h. Adult urinary output should be between 30 and 50 cm^3/h if there is no ongoing hemolysis. If there is hemoglobin and myoglobin in the urine, as indicated by a cherry red to outright black color, urinary output should be pushed to the range of 250 cm^3/h or greater. This is accomplished by using fluids and Mannitol (Astra/Merck Group of Merck & Co., Inc.) as a means of mechanically washing these products from the renal tubules where the sludge tends to accumulate with damaging results. Rapid and adequate resuscitation can minimize or eliminate acute tubular necrosis, which remains associated with virtually 100%[12] mortality in burn patients.

Administration of Mannitol during resuscitation may artificially increase urine output through its diuretic effect, leading to hypovolemia and dehydration. The hematocrit and hemoglobin need to be carefully followed. Mannitol can provide predictable, smooth diuresis, which helps counteract the natural reflex tendency of the kidneys to slow urinary output due to vasoconstriction and the actions of antidiuretic hormone, catecholamines, and aldosterone. Mannitol is also a powerful antioxidant that may have significant benefit in mitigating the effects of free oxygen radical production in the burn wound.

Fluid Resuscitation for Burn Shock

A burn injury sets off a cascade of local events and humoral responses that continue for 48 hours postinjury, resulting in edema formation and fluid losses through the wound. As the burn wound exceeds 15% to 20% TBSA, the fluid losses can exceed the body's compensating mechanisms with circulatory collapse. If distance and time of transport are lengthy, the casualty may suffer burn shock by the time of arrival in the treatment facility. Self-aid or buddy aid will be ineffective. The casualty should receive emergency medical treatment by the trained technician in the evacuation ambulance.

A fluid resuscitation program is designed to overcome the anticipated 24- to 48-hour period of massive fluid shifts, electrolyte derangements, acid base imbalance, edema formation, and fluid and protein losses. More detailed information is found in Exhibit 10-2 and Table 10-2. Replenishing the circulatory volume, using any one of the formulas currently in use, is the means to restoring and maintaining organ perfusion and function. The fluid loss that depletes the circulatory volume is the result of increased capillary micropermeability affecting burned and nonburned tissues.[13] Edema fluid forms at the expense of the vascular and extracellular compartments. Electrolyte concentrations can vary con-

EXHIBIT 10-2

AVERAGE FLUID RESUSCITATION REQUIREMENTS (CM^3 CRYSTALLOID/ KG/% TBSAB)

Burn	Average adult	2
	Average child	3
Burn and Inhalation Injury	Average adult	4
	Average child	5

TBSAB: total body surface area burned

TABLE 10-2

TYPICAL FLUID RESUSCITATION PROGRAMS

Formula	Fluid Composition	Calculated Volume	0-8 Hours	9-24 Hours	24-48 Hours
Carvajal Children	Na 132 mEq/L Cl 109 Lactate 26.1 K 3.8 Glucose 47.5 g/L Albumin 12.5 g/L	5,000 mL/m^2 TBSAB/d[1] + 2,000 mL/m^2 TBSA/d[1] (maintenance fluid)	1/2 vol	1/2 vol	3/4 of day 1 vol
Infants	Na 81 mEq/L Cl 61 HCO$_3$ 20 K 0 Glucose 46.5 g/L Albumin 12.5 g/L		1/2 vol	1/2 vol	
Brooke	Ringers lactate Na 130 Cl 130 Lactate 28 K 4 Ca 3	1.5 mL crystalloid/kg/% TBSAB 0.5 mL colloid/kg/% TBSAB + 2,000 cc D$_5$W	1/2 vol crystalloid and colloid	1/2 vol	1/2 of day 1 vol
Parkland	Ringers lactate D$_5$W + 0.5-2 L	4 cc/kg/% TBSAB (to maintain urine output at 50 cc/h) plasma	1/2 vol	1/2 vol	
Hypertonic Saline	Na 200-300 mEq/L Cl -100 mEq/L HCO$_3$-150 mEq/L	2 cc/kg/% TBSAB (to keep urine output at 30 mL/h [adult] or 1 cc/kg [child])			

Note: For accompanying inhalation injury, add 1-2 cc/kg to resuscitating volume
TBSAB: total body surface area burned
Source: (1) Carvajal HF. Fluid resuscitation of pediatric burn victim: A critical appraisal. *Pediatr Nephrol*. 1994;8:358.

siderably, due in part to large potassium intracellular losses, urinary excretion, and large sodium intracellular and extracellular gains.[14] The currently used burn resuscitation formulas are quite similar in both fluid volume administered and milliequivalent of sodium given when both are calculated at 48 hours postburn.[15] The key component in each formula is the sodium ion; the fluid (free water) is simply the vehicle to deliver the sodium. Depending on the choice of formula, the fluid volumes range from 2 to 5 cm^3/kg/% TBSA. Some formulas use no colloid, others vary in the amount and timing of colloid administration. The role of crystalloid vs colloid resuscitation remains an ongoing debate fueled by ample data and opinion on each side. It is important to note that each formula is merely a guideline for resuscitating the casualty victim. Predictably, the individual response to a prescribed formula may be quite unpredictable and variable, and require corrective adjustment in either direction. None of these formulas reflect evolving trends that add a sophisticated medical resuscitation component to the already existing fluid protocol. The following is a partial listing of components being used or investigated for improved resuscitation, or decreased burn wound edema supplement resuscitation, or decreased burn wound edema and the inflammatory response in the early phase: low molecular weight dextran, fresh frozen plasma, pentastoid, Mannitol, ibuprofen, cimetidine, and vitamins C and E.[15-24]

Contrary to the opinion of some clinicians,[25-29] excess edema postresuscitation can have severe, long ranging consequences. Organ systems such as brain, lungs, and heart do not function well under the stress of severe edema. Circulation to an edematous extremity can be compromised with possible dire consequences. Early escharotomy is indicated when massive edema is anticipated. Early mobility and rehabilitation may be unsafe, if not nearly impossible, when edema impairs joint motion. Loss of dexterity in edematous hands and digits can further compound the person's feeling of helplessness following injury, leading to a vicious circle of decreased use, joint stiffness, contracture, tendon shortening, and long term disability. Therefore, it is imperative to adequately resuscitate but to do so with minimal edema formation.

The Nonhealing Burn Wound

The surgical goal is to expedite wound closure. Estimation of burn wound depth is an important clinical judgment because it determines proper treatment. Superficial and full thickness char burns can be easily determined. The intermediate or partial thickness burn is difficult to assess. A partial thickness burn wound may vary in depth within a small area, thus, healing may be patchy or delayed. A burn wound failing to heal in 18 to 21 days typically requires skin grafting because the final functional and cosmetic result of a skin graft is superior to the poor quality skin covering of a delayed healing wound. This is especially true in the pediatric age group.

Dressings impregnated with hormones or other factors will soon be available to stimulate improved healing of superficial and intermediate burn wounds. Early excision and grafting of the full thickness wound will remain the standard of burn care in most cases. Although still unproved in controlled studies, early excision is certainly based on sound principles of general surgical wound management and has clinically proven effective[30-35] in diminishing morbidity in the burn casualty.

Burn Wound Excision

The estimated depth of the burn wound often determines the choice of excision technique: tangential, sequential tangential, or fascial excision. Superficial wounds are best excised with sequential passes of the dermatome until viable tissue is exposed. Charring burns deep into subcutaneous fat are excised in segments to the fascia.

Tangential Excision

Dermal burns are debrided with multiple passes of the debridement instrument until viable tissue is reached; this is known as sequential tangential excision. This method of debridement allows maxi-

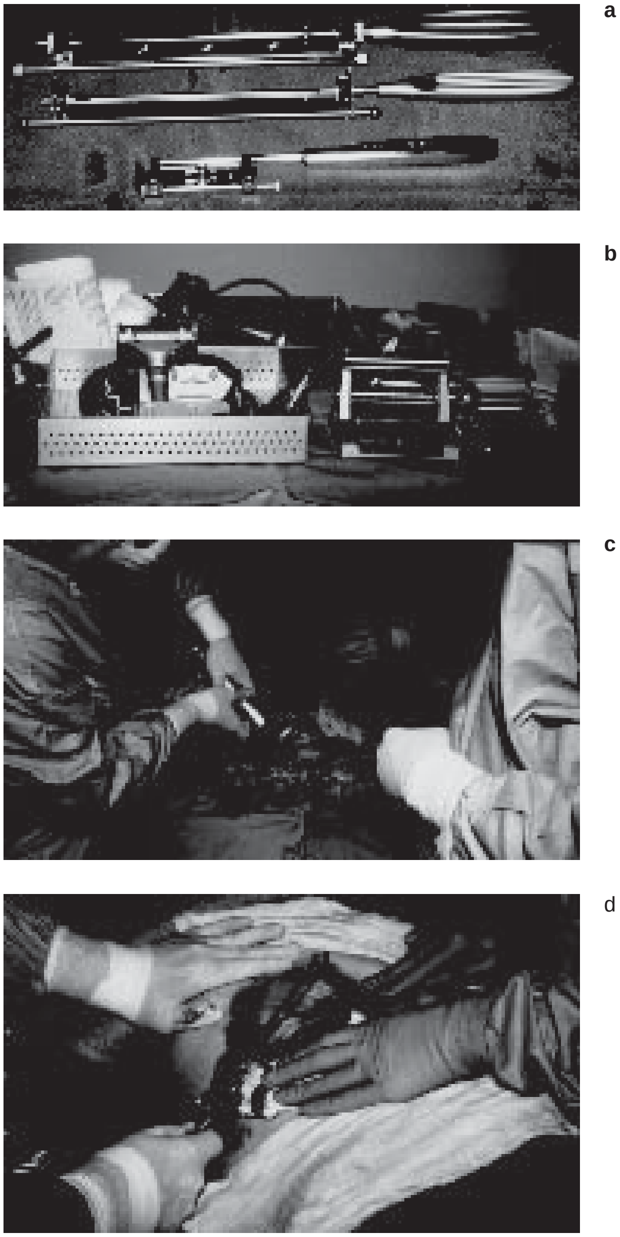


Fig. 10-13. Instruments for graft harvest or debridement. (**a**) Example of manual dermatome for skin debridement or graft harvest. (**b**) Example of power dermatome. (**c**) Skin debridement in progress using power instrument. (**d**) Skin debridement in progress using manual instrument.

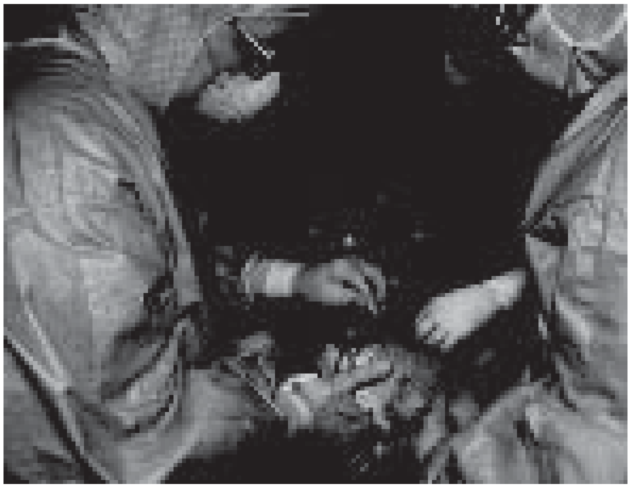


Fig. 10-14. Brisk punctate bleeding from viable dermis follows tangential debridement.

mum preservation of viable dermis, which ultimately leads to better long term results in the quality of the healed grafted skin.

There are any number of instruments available for wound debridement, some manual, others electrical or air driven (Figure 10-13). The technique of debridement involves setting the dermatome somewhere in the range of 0.008 to 0.012 in. for excising partial thickness burns or cutting up to 0.030 in. for full thickness burns. Areas requiring intricate or delicate work, such as digit web spaces, are best approached using small instrumentation with shallow settings in multiple passes.

Areas that have been adequately debrided show brisk punctate bleeding in healthy white dermis (Figure 10-14). Poor bleeding in grayish appearing dermis indicates inadequate debridement, which requires repeat passes with the instrument. As debridement progresses deeper into and through the dermis, more fat appears and capillary bleeding gives way to brisker flow from arterioles and veins.

Hemostasis in the debrided wound is obtained using pinpoint electrocautery on larger vessels and dilute epinephrine soaked lap pads on the diffuse capillary bleeding bed. Local pressure and temporary elastic wraps can assist bleeding control. Topical epinephrine solutions in concentrations ranging from 1:10,000 to 1:100,000 can be safely used with few systemic effects in acutely burned patients. Topical thrombin and collagen have also been used for this purpose.

Blood loss from the debrided wound bed can be profuse and can exceed 5% of blood volume per 1% body area debrided. Blood loss of this magnitude requires frequent monitoring of hemoglobin/hematocrit, platelets and coagulation factors, casualty temperature, blood pressure, and urine output. In a well prepared and monitored casualty it is possible for multiple surgical burn teams to rapidly excise and cover a 50% body surface area (BSA) burn in a single 2-hour session. Less experienced teams should limit excisions to under 30% surface area, or 2 hours time.

Excision to Fascia

Deep burns through dermis well into the subcutaneous tissue are best excised with an electrocautery knife to the fascial plane. The excision at this level can proceed rapidly with less blood loss. On extremity burns blood loss can be minimized by the use of tourniquets. When using a tourniquet, the extremity is first exsanguinated (the limb is tightly circumferentially wrapped from distal to proximal to empty the capillary, arterial, and venous circulation) and excision proceeds in a rapid manner along the fascial planes removing all eschar with the underlying subcutaneous tissue. This can lead to major tissue losses, especially in obese casualties with subsequent significant cosmetic defects. To obtain optimal functional and cosmetic results, it is important to obtain nearly complete hemostasis to prevent graft loss.

Graft adherence ("take" or attachment) on fascia is generally better than onto subcutaneous fat. This better graft take, however, comes at a price with inferior cosmetic appearance, increased edema in distal extremities, and, at times, decreased sensation.

Enzymatic Debridement

Enzymatic debridement of the burn wound has multiple advantages, such as more complete and selective debridement of the burn eschar, while at the same time being nontoxic and nontraumatic to normal tissue. Debridement of a deep hand burn is an ideal use of this methodology. There is a risk of promoting sepsis with enzymatic agents, thus their application should be restricted to under 10% of the TBSA. Metal ions such as those found in soaps and antimicrobial agents tend to inactivate enzymes, and therefore, should not be used together.

The enzymatic debridement procedure is as follows.

1. Mechanically debride affected areas of all loose tissue.
2. Wash and rinse area thoroughly.

3. Apply a uniform coating of debridement enzymes such as Accuzyme.
4. Cover area with occlusive dressing. Hands are easily dressed with oversized surgical gloves.
5. Repeat application of enzyme every 4 to 6 hours until punctate bleeding is observed (two or more applications are typical).
6. Do not exceed enzyme application over 10% of TBSA to avoid risk of burn wound sepsis.
7. Chemical escharotomies of circumferentially burned limbs can be performed using strips of the enzyme paste along the length of the burn. If the limb is acutely ischemic, then standard escharotomies should be performed for immediate decompression.

Donor Site Selection

Donor sites are selected on the basis of location, skin thickness, burn size, function, and cosmetics. The face, hands, and feet are rarely used for donor sites, whereas the scalp, thigh, abdomen, and buttocks are frequent choices. If the burn is small, the site selected is usually determined by future cosmetic concerns. The scalp, usually covered by hair, and the buttocks, covered by clothing, are therefore, frequently chosen for donor sites. In the massively burned casualty the scalp is an ideal donor site since it heals rapidly and can be reharvested repeatedly at 7- to 10-day intervals with little risk of alopecia.

The thickness of the skin influences the donor site; for example, the dorsum of the hand may be less than 0.010 in., the back greater than 0.030 in. The back, buttocks, and thighs are often chosen as donor sites because of their thickness and ease of harvesting. Surface contour and subcutaneous adipose deposits can greatly influence the ability to harvest quality grafts and thus have a negative influence on site selection. In body areas such as the ribs or scalp, subcutaneous injection of warm saline improves the contour for skin harvest.

Skin Grafts

Split Thickness Skin Grafts

A split thickness skin graft (STSG) is typically harvested in a range from 0.007 to 0.012 in. using either a manual or powered dermatome. The advantages of using power dermatomes (Figure 10-15) are in the speed, uniformity, and precision of skin harvest. These delicate instruments have a low tolerance of abuse and a high frequency of expensive repair.

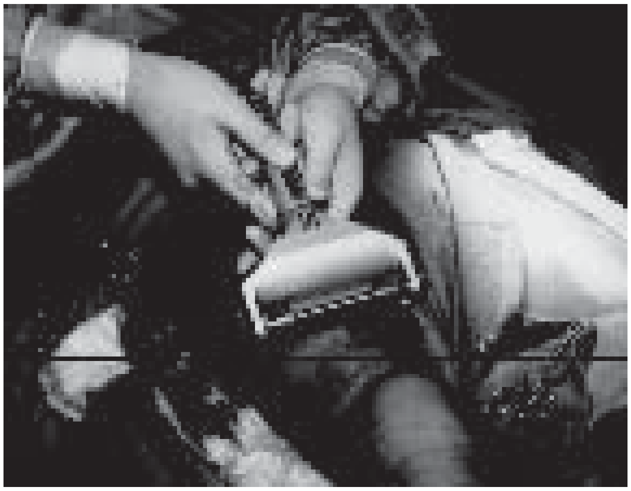


Fig. 10-15. Harvesting a split thickness skin graft using a Paget electric dermatome.

Sheet Skin Grafts

Areas of burn graft where an optimal functional or cosmetic result is desirous should be covered with sheet skin grafts. A sheet graft is harvested as noted above and is applied directly to the graft bed without first being meshed as described below. The sheet graft does not have perforations or incisions and is not expanded, thereby reducing scarring. Examples of areas best served by sheet grafting include hands, face, and neck. Availability of adequate donor sites affects how much grafting can be accomplished with sheet grafts.

When blood or serum collect under sheet grafts, the grafts may lift off the bed, resulting in graft loss. Extra time needs to be devoted to obtaining adequate hemostasis, otherwise the grafts slough over fluid collections, which will result in a cosmetic appearance worse than a mesh graft. Any large fluid collection can threaten the entire graft take and needs to be locally drained.

Meshed Skin Grafts

The instrumentation to accurately and reproducibly mesh skin, developed by Dr. Tanner, helped to revolutionize burn care by providing a method of skin coverage for the massively burned casualty. Skin expansion allows for greater graft coverage from limited donor sites. The surgeon has a choice of

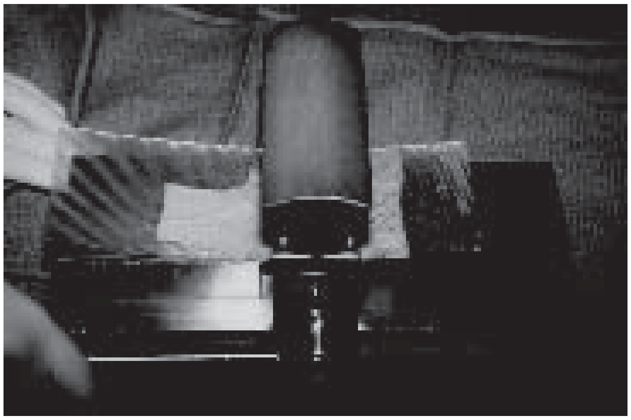


Fig. 10-16. The Zimmer mesher, which uses fixed ratio carriers from 1.5:1 to 9:1.

multiple expansion ratios ranging from 1.5:1 to 9:1. As the expansion ratio increases, the quality and cosmesis of the skin decreases as the healing time for the graft interstices increases. As a general rule, most coverage is accomplished with mesh ratio of 3:1 or less.

There are two basic types of mesh instrumentation. One instrument, the Zimmer mesher (Figure 10-16), uses carriers for the skin that are chosen according to the desired ratio. The other instrument, the Bioplasty mesher, (Figure 10-17) has a preset ratio requiring no skin carriers. Instead, the skin is fed directly into the rollers. The advantage of the Bioplasty mesher is that harvested skin of any shape and length can be fed into it and meshed without seams, which are normally present when carriers are joined to make longer lengths. Fewer seams result in fewer hypertrophic scars, which decreases rehabilitation time. Faster meshing of skin and lower cost make this dermatome more practical to use.

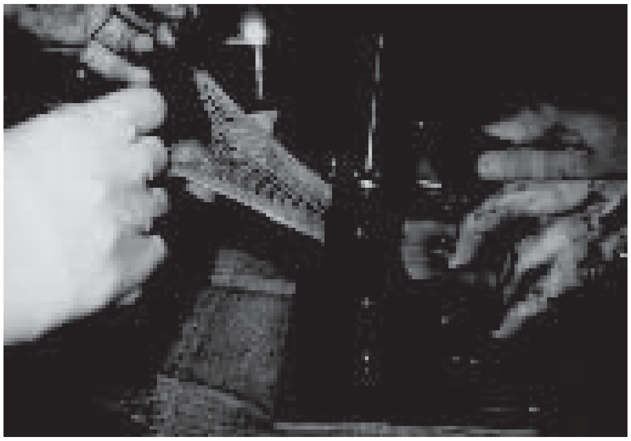


Fig. 10-17. The Bioplasty mesher has a fixed mesh ratio built into the instrument, which cannot be changed. No carriers are required. Bioplasty meshes can be obtained with preset ratios from 1:1 up to 8:1.

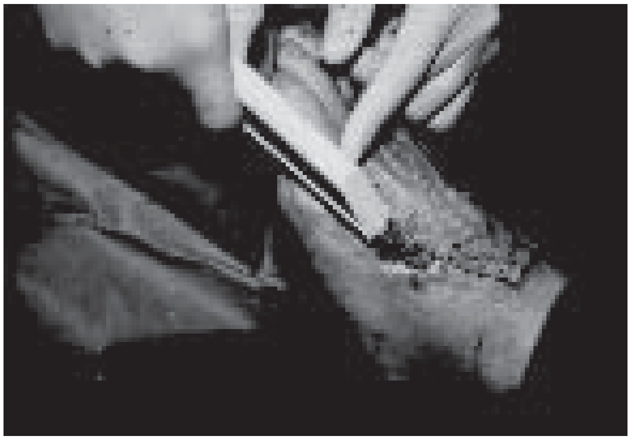


Fig. 10-18. Skin grafts are rapidly secured using surgical staples, saving considerable time over the traditional hand sewn method.

Skin graft adherence is a two-part adhesion response beginning with early fibrin bonding when the graft is set on the bed. This is followed by fibrocapillary ingrowth in the ensuing 72 hours. Although tenuous and easily sheared at this time, the viability of a graft is assured by this capillary inosculation. More grafts are lost at this phase through motion and shearing than infection.

Proper management during the immediate postoperative period can significantly affect the final outcome. Graft movement is the first cause of graft failure; infection is the second. Securing the graft to the bed is important because graft motion or shift is the greatest cause of graft failure. Before the advent of staples, all grafts were sewn in place, requiring hundreds of individually placed stitches. Now large grafts can be secured in a fraction of the time with surgical staples (Figure 10-18). In pediatric patients, staple removal is traumatic and it may be wise to secure grafts with absorbable suture or Steri-strips (Johnson & Johnson Medical Inc.). Absorbable staples and fibrin glue show future promise. Splints incorporated into the final dressing help maintain desired position and help prevent graft loss due to motion, especially over joints. Proper splinting techniques are reviewed elsewhere in this text.

To minimize infection, the new graft requires proper wound management. Depending on a par-

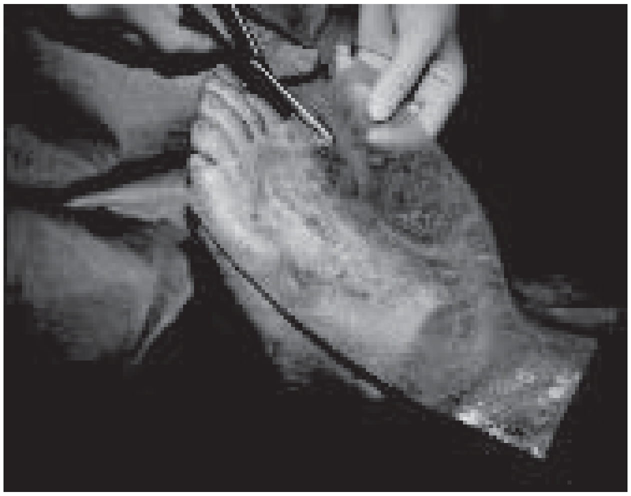


Fig. 10-19. A nonadherent dressing may be cut to size to cover a fresh skin graft.

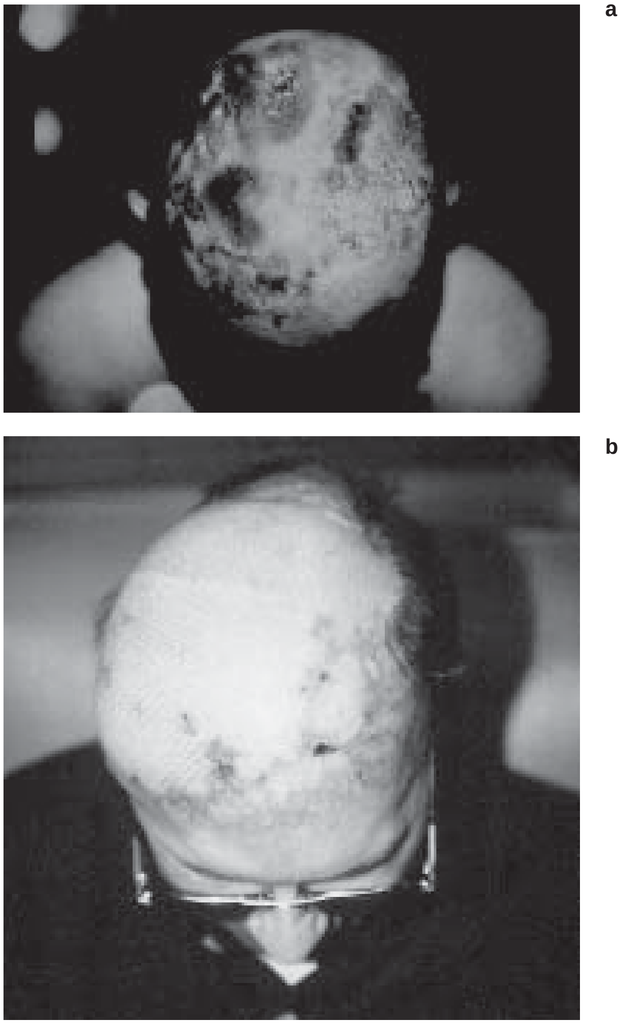


Fig. 10-20. Care of electrical injury. (**a**) Electrical burn of scalp from high voltage contact. Note exposed skull. (**b**) Closure with latissimus dorsi free flap and skin graft six months later.

ticular burn center's protocol, skin grafts are treated in an open or closed method. Centers using the closed method apply a nonadherent contact layer to the graft. Tegapore, N-ter-face, and Adaptic (Johnson & Johnson Medical Inc.) are some of the dressings available for this purpose (Figure 10-19). Small burns are dressed with antibiotic soaked gauze, dry gauze, and elastic compression. Small irrigation tubes may be used in the dressing to maintain a moist antimicrobial environment. Either a 2% cerium nitrate solution or an antibiotic solution containing Kanamycin, Bacitracin, and Polymyxin (Burroughs Wellcome Co.) are used. Irrigation is continued until the first dressing change at 3 to 4 days for meshed grafts and 24 hours for sheet grafts. The dressings are kept moist but not wet.

At the first dressing change, the now adherent dressing can be left in place provided there are no signs of suppuration. This layer is easily removed several days later, as the graft interstices fill in, by either soaking the dressing just prior to removal, or applying silver sulfadiazine cream during the previous day's dressing. Grafts showing signs of infection can often be salvaged by removing all dressings, gently irrigating and debriding with once- or twice-a-day dressing changes.

Free Flap

Burns that create large fourth degree wounds (injury to muscle and bone) can be covered by flaps. Traditionally, these have been flaps rotated on a pedicle base into the area of defect. Occasionally there is no way to rotate a flap into a wound which is also unsuitable for grafting. The more recently developed method of free flap transfer to a tissue defect has had excellent results. The free flap has greatly facilitated coverage of the difficult "blow out," electrical-type wounds of the scalp, joint spaces, palms of hands, and soles of feet (Figures 10-20 and 10-21).

Cultured Autologous Kertinocite (Epidermal) Grafts

Human epithelial cells can now be grown in cultures with techniques developed by Rheinwald and

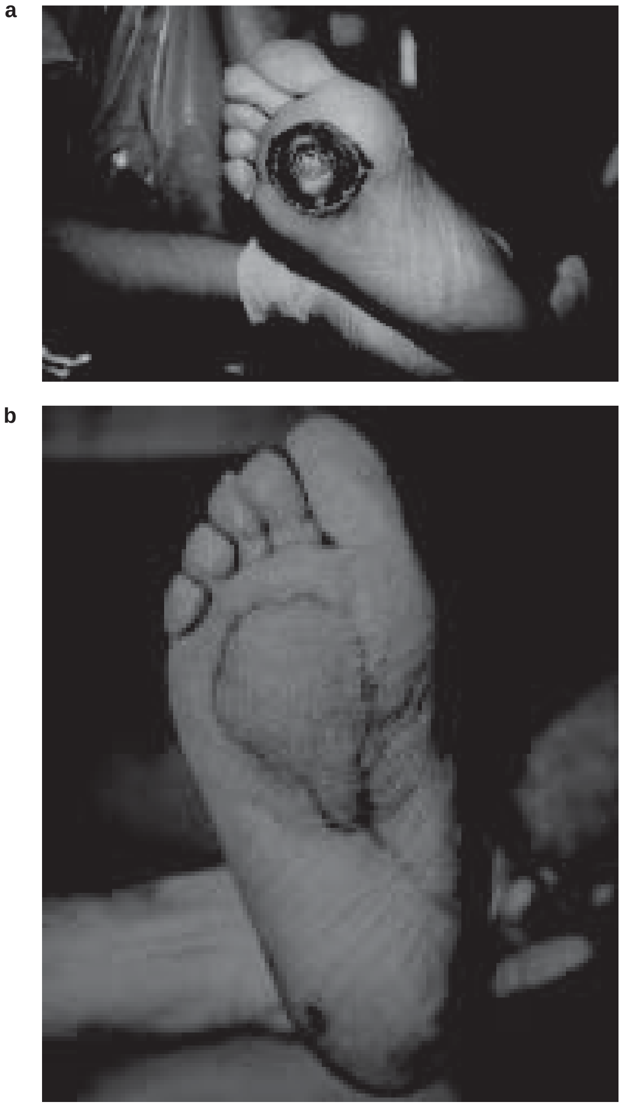


Fig. 10-21. Casualty who sustained an 8,000-V contact burn to the scalp (see Fig. 10-20) with large soft tissue and bone necrosis. (**a**) A deep necrosing wound to the foot with loss of foot tissue and bone. (**b**) Free flap coverage to the sole of the foot provided a substantial graft, which tolerates ambulation and avoided leg amputation.

Green,[37] and modified by Pittelkow and Scott.[38] The technique is a multistep process starting with the harvesting of a small, several-centimeter square piece of thin skin from an unburned site. The epithelial cells are enzymatically cleaved and separated, then placed in a serum-free culture medium where, under ideal conditions, rapid growth is achieved. Keratinocyte sheets, approximately 7 to 10 cell layers thick, are fragile and thin, with the consistency of wet tissue paper.

Careful preparation of the wound bed is of utmost importance prior to grafting with cultured grafts. These grafts will not reliably take on anything but a freshly excised bed. Graft adherence initially is through fibrin adhesion. A backing such as Vaseline gauze, stapled to the wound or meshed cadaver grafts stretched tight over the cultured grafts can be used to assist adherence.

Another approach to providing a better quality skin is the development of an artificial dermis. The product invented by Burke and Yannas is composed of bovine collagen fibers bonded to chondroitin-6-sulfate, a component of shark cartilage.[39] Ideally, thin epidermal grafts heal to this artificial dermis, resulting in a closed wound. A multicenter controlled study of this dermis in general showed favorable results, concluding that the healed artificial dermis covered with thin epidermal graft is essentially equivalent to standard skin grafts but with faster healing donor sites.[39]

Casualties who are grafted with cultured epithelium do not develop hair, sweat or oil glands, or proper sensation. The epidermis permanently remains extremely fragile and requires extraordinary protection from exposure to sun, chemicals, and trauma. Rehabilitation is exceptionally complex. The epithelium tolerates exercise, splinting, and external vascular supports poorly. These casualties may return to have the cultured grafts replaced with autografts as the donor sites become healed enough for reharvesting. The ideal artificial skin replacement has yet to be developed.

Biological Dressings

The surgical goal in burn care is to debride nonviable tissue and provide permanent wound coverage. Biological dressings are designed to function in a similar manner as their more expensive natural counterpart, cadaver skin. There are many biological dressings available, each with its own properties, but none is a true skin replacement. Biological dressings provide temporary coverage and give time for adequate donor sites to become available for repeated graft harvest. This coverage is important in the overall physiological well being of the burn casualty as it provides for better wound and pain management, helps decrease metabolic rate, reduces fluid losses through the wound, and suppresses the growth of granulation tissue.

The ideal biologic dressing would function as natural skin and come prepackaged off the shelf, ready to apply and perform as a complete skin replacement on a permanent basis. Synthetic dressings are laboratory designed to mimic their biologic counterpart, skin. Since these act as biologic dressings, for the purpose of this discussion, they will be reviewed together with the true biologic dressings. A satisfactory biologic dressing undergoes the same bonding process as skin grafts. Most importantly, when adherence is achieved, the wound gains resistance to infection.

Homograft (also known as Allograft) Skin: Human cadaver skin is commonly used and is the standard against which other biologic and synthetic dressings are measured. Banked frozen human skin is becoming more widely available but is inferior to its fresh counterpart. In the massively burned casualty, no currently available biologic dressing offers the distinct advantages of fresh homograft in providing large burn wound coverage. Improved salvage of these casualties is based on early aggressive burn wound excision and coverage with homograft followed by sequential replacement with autologous skin.

Fresh homograft adherence is similar to autograft take with adherence and capillary ingrowth. A burn casualty with greater than 50% TBSA injury is, to a varying degree, autoimmunosuppressed. This state of immunosuppression can result in slowing of the natural rejection process, especially when the homograft skin is ABO compatible. Aside from providing excised wound coverage, homografts can also be used as onlay grafts to protect thin, widely expanded autografts, or cultured keratinocytes, providing protection to the underlying grafts.

Homograft rejection occurs anywhere from 14 to more than 60 days after the grafting, depending on the casualty's immune status. Replacement homografts tend to reject at a faster rate than original grafts. The rejection process affects the wound bed and may negatively influence further grafting. It is advisable to completely excise the bed prior to grafting.

Availability, cost, and the potential risk of viral transmission are the major drawbacks of fresh homograft. Procurement, preparation, and storage costs are relatively high. In spite of testing, there remains the possibility of viral transmission, including hepatitis, cytomegalovirus, and human immunodeficiency virus (HIV). There is at least one known case of HIV transmission involving autograft from England in 1987.[40,41] Amnion is used for temporary coverage in underdeveloped countries, but not in the United States, and carries the same infectious risks.[42,43]

Because the graft take is expected to be prolonged, the graft area is immobilized postoperatively in a similar method as autografts. Rehabilitation of cadaver grafted limbs can commence in 5 to 7 days.

Heterograft and Synthetic Dressings

Porcine skin heterograft is currently used in this country. It is a nonviable graft with limited usefulness in short term, temporary wound coverage. It has no advantage over homograft and has numerous disadvantages. If used, range-of-motion exercises need not be curtailed.

Numerous synthetic dressings are currently available to the burn clinician, with many more to come in the future (Figure 10-22). Fortunately, there is this large choice of products because there is no one ideal dressing. Also, what works well at a burn center may not work well in a combat zone. Some dressings have been designed for a specific purpose while others are adapted to a wide variety of clinical applications. The choice of a particular dressing is usually based on a mixture of tradition, art, and science.

The synthetic dressings range from the simple transparent polyurethane or polyurethane membranes (Tegaderm, Opsite [Johnson & Johnson]) to a more complicated bilaminate membrane, Biobrane (Winthrop Consumer Products). This dressing is composed of collagen peptides bound to a silicone nylon mesh. To function properly this dressing must be bound to the tissue by ingrowth into the collagen network. Biobrane has been shown to perform effectively on partial thickness burns or excised wound beds.[44] It is contraindicated for use over full thickness burns. Because it binds to the tissues, range-of-motion exercises are temporarily restricted.

Film dressings such as Tegaderm or Opsite work well to relieve pain and protect skin donor sites.

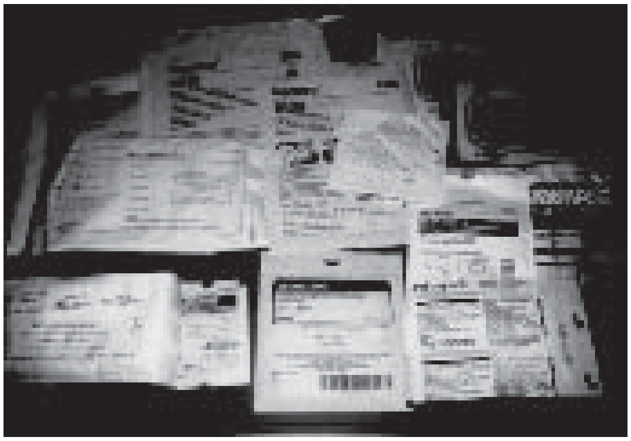


Fig. 10-22. Some of the numerous synthetic dressings available for use in graft and wound care.

These dressings also decrease pain, protect, and decrease fluid loss in small, isolated minor burns treated in the outpatient clinic. There is no restriction in range-of-motion exercises. Partial thickness burns are covered with nonadherent dressings such as Adaptic, Frastec, N-ter-face, or Tegapore (Winthrop Consumer Products), which separate easily, when adherence is not desired. Antimicrobial ointment helps secure the dressing while an absorbent layer of gauze is applied, and can generally be used for small burns.

Various first generation hydrocolloid dressings such as Duoderm (ER Squibb & Sons) are now available and have been used on partial thickness burns and on donor sites. This dressing performs poorly on full thickness burns.[45] Pain under a hydrocolloid dressing is unpredictable. Casualties complain about the drainage as this dressing becomes saturated with exudate from the wound. The calcium alginate dressing Sorbisan (Dow Hickman Pharmaceuticals Inc.) has a similar drawback. Exercise programs continue with these dressings, but for a foot wound, exudate accumulates in the shoe and becomes messy.

Donor Sites

As burn size increases, the donor site takes on added importance. The donor site should be considered the equivalent to a partial thickness burn, which must heal like its burn counterpart. Rapid healing of the donor site is imperative because of the numerous repeat harvestings necessary to close a massive burn. The scalp is probably the ideal donor site since it heals so rapidly and has few associated complications. A scalp donor site covered with a clear polyurethane dressing can be expected to heal in 5 to 7 days for a graft harvested at 0.008 to 0.010 in. (Figure 10-23). As a general rule, the farther the donor site is from the heart, the longer it will take to heal.

Dressing the donor site is done to help in decreasing pain and promoting rapid healing. There is little pain under the clear film dressings (Tegaderm [3M Health Care], Opsite [Johnson & Johnson], Omniderm [Jobst]) when compared to fine mesh gauze, scarlet red, or open treatment. The glue of the clear film dressings does not adhere to the actual donor site, thus a several-centimeter rim of normal skin surrounding the donor site is required for dressing

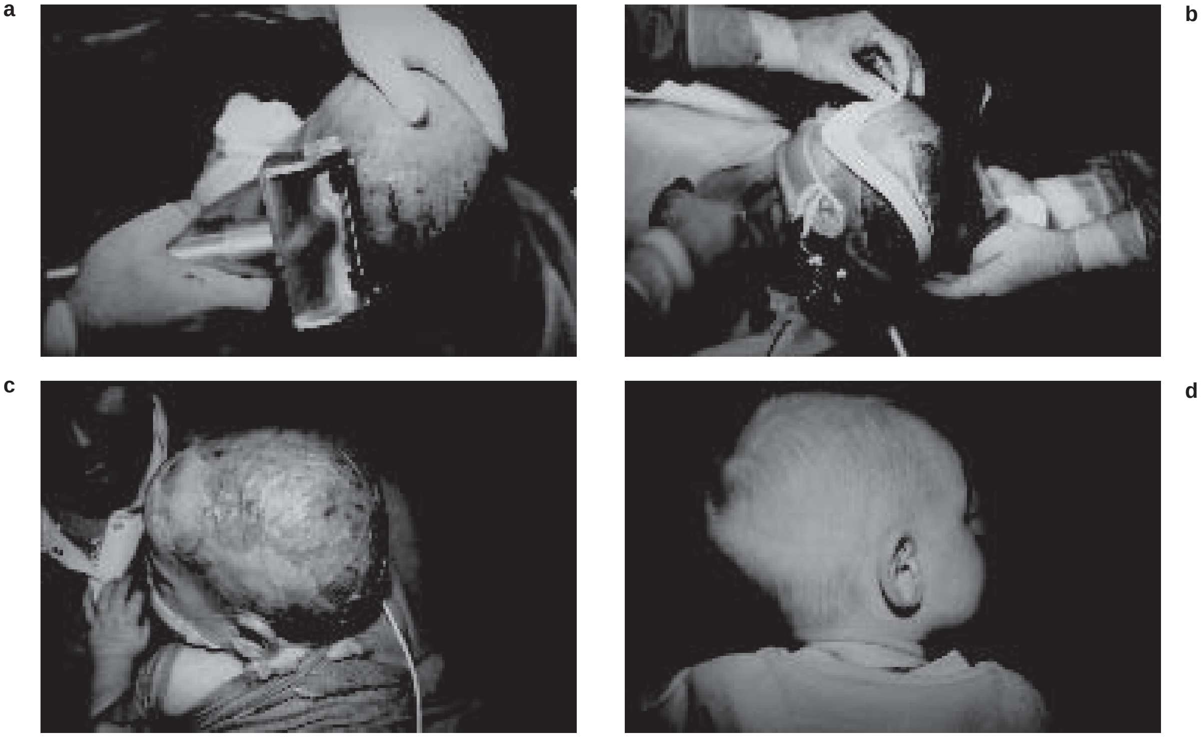


Fig. 10-23. Use and healing of scalp donor. (**a**) Harvesting scalp in a child at 0.008 inch. (**b**) Covering donor site with polyurethane dressing (Tegaderm). (**c**) Appearance of scalp covered with dressing in early postoperative phase. (**d**) Same scalp donor site approximately one month postharvest showing hair regrowth.

adherence. For donor sites exceeding the size of the largest dressing, multiple dressings can be patched one to another to obtain complete coverage.

The donor site becomes more of a problem as it is harvested adjacent to or between burn areas, a frequent occurrence in the massive burn casualty. If this donor site is covered with a clear film dressing it will rapidly become infected. It is preferable to treat thin donor sites in a more open manner with a nonadherent dressing such as Adaptic (Johnson & Johnson) and a topical antimicrobial similar to the way the adjacent burns are treated.

The clear film dressings are gas and water vapor permeable, but not so to the serum or blood that often collects from the donor site. The pressure of the fluid buildup can disrupt the dressing integrity along the dependent edge with potential for bacterial contamination. Aspiration of fluid and patching of the dressing can minimize this problem.

Donor sites can be a serious problem for children and the elderly. It is difficult to maintain a dressing on children. At these ages the skin is much thinner, and, therefore, the harvested grafts are relatively thicker in terms of amount of dermis removed. The donor site becomes a wound as deep as the original burn and is often very slow to heal. Children are prone to hypertrophy of their donor sites with the end result a cosmetic appearance sometimes worse than the grafted sites.

To avoid problems with the nonhealing donor site in the elderly, the surgeon can harvest extra skin, expand it 3:1, and regraft the donor sites. Excellent results have been obtained using this technique.[46]

Healed donor sites can have problems with blistering, usually self-limited, and pigmentation changes. To minimize splotchy pigmentation, sun exposure should be avoided for a year or more. External vascular support garments should be used on hypertrophic donor sites as well as on healed burn grafts.

Goals of Rehabilitation Management

The optimal rehabilitation goals after a burn injury include: (*a*) healed burn wounds, grafts, and donor areas. (Ideal results include wounds that are durable, soft, supple, flat, properly colored, pain and edema free, and able to tolerate active duty and work.) (*b*) maximal possible status for strength, endurance, AROM, fine motor dexterity and coordination; and (*c*) independent self-care for return to active duty. Optimal recovery is not always possible. In these circumstances the goals are that the casualty is: (*a*) managing epithelial sensory, vascular, and pigment changes through in depth knowledge of protective safety interventions; (*b*) managing edema; (*c*) continuing prolonged stretching of contractures to maintain acceptable motion and appearance; (*d*) continuing aerobic exercise, attempting improved cardiopulmonary status and endurance; (*e*) using appropriate protection from exposure to extremes of heat or cold or trauma; (*f*) learning desensitization and management of itching or hypersensitivity; and (*g*) coping with posttraumatic stress symptoms and permanently changed body appearance.

MANAGEMENT OF BURN CASUALTIES

For purposes of discussion only, burn care can be divided into three phases: (1) acute, (2) immobilization, and (3) wound maturation phases. The acute, pregrafting or early phase of treatment begins when the casualty's burn occurs and is followed by at-the-scene care from the battlefield medic, emergency treatment during transportation to a battalion aid station, evacuation from a mobile army surgical hospital, and finally, hospital admission for wound care followed by reconditioning and rehabilitation for return to duty.[47] In large burns the acute phase consists of the initial 72 hours of resuscitation and shock followed by up to 3 weeks of wound and patient management to achieve primary healing if the wound is superficial. A longer time period is anticipated for grafting when the wound is deeper or greater than 20% TBSA. This phase continues through the healing of partial thickness tissue injury on through debridement or preparation for grafting in full thickness tissue destruction. For chemical injuries, the rehabilitation treatment after the initial flushing is the same as for thermal heat injuries. There are no limits to AROM. Ambulation is encouraged as soon as the casualty is medically stable. Frostbite care, however, in the acute phase is slightly different because the cell is temporarily crystallized and caution is used to prevent any pressure on the damaged tissue until all areas thaw. In addition, frostbite blisters are kept intact, which means active motion must be done very cautiously in bed (Figure 10-24). In the case of an electrical injury, AROM is continued from admission through debridement, even though tendons may be exposed and amputation of a distal extremity is anticipated. Distal function in the electrical injury is often deceptive. The often observed delayed microvascular

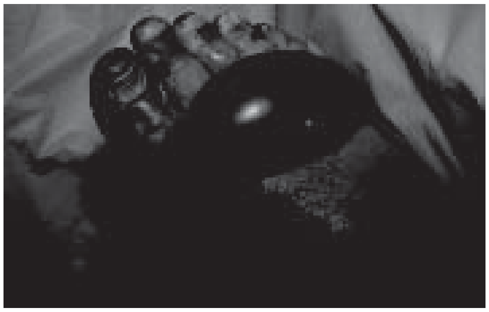


Fig. 10-24. Intact frostbite blister that complicates wound management, exercise, and ambulation.

thromboses in the extremity results in gradually increasing demarcation of necrosis. However, distal function is maintained as well as possible until need for amputation has been determined.

The grafting or immobilization phase of treatment begins after the casualty is taken to surgery for grafting and continues until the graft is adherent or has taken and the patient is allowed to be up walking. Care and rehabilitation for all four types of burn injury are similar during immobilization for graft adherence.

The wound maturation phase of treatment begins when the wound is healed and ends when the wounds are mature and prosthetic fitting, if necessary, is completed and when all reconstructive procedures are completed. The wound maturation phase includes the shorter inpatient recovery time, during which open areas become small enough for safe discharge from the hospital and the longer convalescent period, which lasts until all possible rehabilitative and surgical interventions have been performed and all grafts, scars, and donor areas are mature. The long final phase of wound maturation may begin in a rehabilitation center or take place as an outpatient in the home or both.

In actuality, all of these phases of healing overlap because an injury is rarely of a single depth, and varying parts of the body are also at different stages. However, there are general principles of rehabilitation and convalescent care that apply to all phases of healing. These general principles involve exercise, elevated positioning, orthotic management, functional activity, adaptive equipment, and psychosocial adjustment. Therefore, generic comments concerning each of these topics will be discussed initially, with details presented under each phase of wound healing.

Active exercise maintains range-of-motion, maximizes strength, minimizes edema, maintains endurance, and improves circulation, thereby speeding wound healing. Passive motion maintains range-of-motion. Proper and varied positioning decreases edema; minimizes contracture development; and prevents dislocations, neuropathies, decubiti, and complications of bed rest such as pneumonia or phlebitis. Orthoses help decrease edema, eliminate or minimize contractures, immobilize grafts for healing, increase functional use of an extremity, correct a deformity, speed healing, protect healing tissue, and modify scar deposition. Functional activity, with adaptive equipment, if needed, improves range-of-motion, fine motor dexterity, and overall endurance. It increases feelings of self-reliance, improves self-esteem, and hastens discharge from the hospital to a less supervised setting. Psychosocial adjustment includes counseling to reduce the initial shock; fear; anxiety; and symptoms, such as sleep disturbance, disturbing dreams, appetite disturbance, difficulty falling asleep, or frequent awakening during sleep. Later counseling provides interventions for complaints of too much or too little sleep, feelings of estrangement or detachment from others, recurrent intrusive memories of the event, memory impairment, difficulty concentrating, reluctance to accept a changed body image, exaggerated focus on pain or itching, decreased interest in sex, sensitivity to loud noises or other cues related to the accident, as well as irritability and fear of returning to active duty or work. Premorbid drug or alcohol abuse is best managed in an outpatient setting, during the wound maturation phase of care. Depression, anger, grieving, and changed sexual functioning are often other focuses of outpatient counseling.

Acute Phase of Healing

The goals in the acute phase of wound healing are to provide analgesia; assist wound closure; prevent infection; modify edema; maintain and improve joint and skin mobility, and improve strength and endurance; facilitate casualty and family participation in therapeutic procedures; improve self-care; achieve independent self-feeding, personal hygiene, use of phone, and writing; increase sense of security, competence, and self-worth; and provide appropriate outlets for anger.

Wound and Skin Care and Nursing During Acute Phase

The potential for infection related to the loss of skin can be a life threatening event for the burn casualty. Meticulous wound care is crucial. On admis-

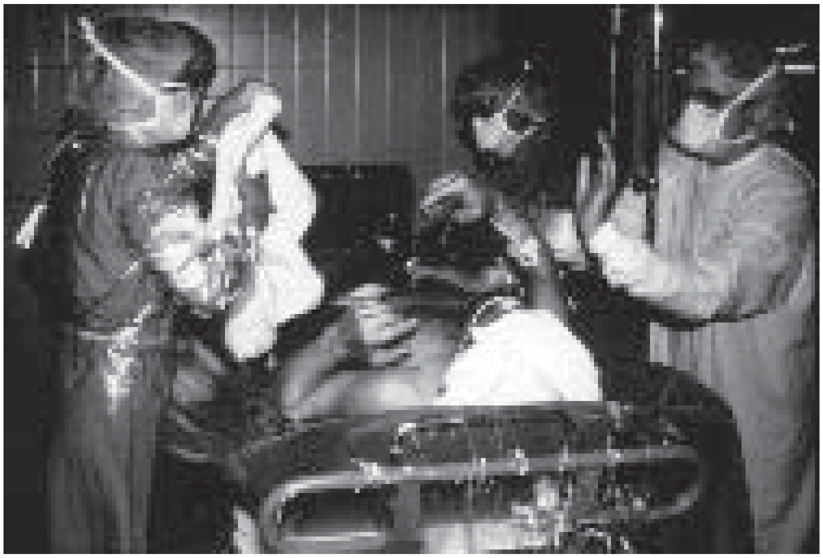


Fig. 10-25. Range-of-motion exercises, unimpeded by dressings during wound cleansing.

sion all areas of the body should be exposed and inspected to determine the extent and depth of the burn injury. A complete head-to-toe secondary survey is also made at this time to ensure all associated injuries have been identified and are being managed appropriately. During this process, which involves wound cleansing and dressing procedures, universal blood and body fluid precautions should be used. This includes, but is not restricted to, the use of plastic aprons, gloves, hats, masks, and protective eye wear. The use of plastic aprons prevents contamination of scrubs with body fluids and also prevents cross contamination to other casualties.

The purpose of wound cleansing is fourfold: it (1) provides a total inspection of the burn wound to examine for any signs or symptoms of infection; (2) allows for cleansing and gentle debridement of the burn wound; (3) provides the opportunity for the cleansing of unburned areas and examination of areas for other trauma and skin redness or breakdown[48]; and (4) encourages unimpeded range-of-motion to the affected areas and complete evaluation of the casualty's range of motion (Figure 10-25).

Wound cleansing can be accomplished through a variety of methods, including use of a Hubbard tank or tub that may contain plain tap water or sodium hypochlorite solution with a ratio of 1:200 (1 part hypochlorite to 200 parts water). Casualties may be suspended over a tub and showered, a commercially available shower cart can be used, or a bedside basin can be used (Figure 10-26). Mortality, incidence of positive blood cultures, and length of hospital stay for patients treated with immersion or rinsing has been studied, and no statistically significant differences were found.[49] Time honored submersion in the Hubbard tank can provide reasonably comfortable soaking to remove dressings and a warm, gravity relieved place for exercise.[50] Its disadvantages include wound contamination from contact with intestinal bacteria that multiply in the bath water (avoided by water rinse over a tub or in bed); edema, when a body part is held dependent in the warm water; chilling if total body is exposed exiting the tub; frightening exposure, especially to children; increased itching from edema and drying; and rebound stiffness one hour after the immersion.

Debridement to remove devitalized tissue and foreign bodies is frequently done in the operating room under anesthesia. Épluchage, the removal of devitalized burned tissue by sharp dissection performed serially with premedication for pain,[52] is reserved for very large TBSA burns. Wounds are usually cleansed at least daily. Thorough cleansing can be accomplished by using a soft gauze sponge, and some burn centers may choose to use a mild antibacterial cleansing agent such as chlorhexidine (Hibiclens) (Exidine) or dilute povidone-iodine (Betadine). Care should be taken not to scrub the wound as this can traumatize damaged tissue possibly resulting in conversion to a deeper wound as well as increased pain for the casualty. Intact blisters should be left intact when they provide a physiologic protective layer over the injured part. However, in the case of chemical burns, the blisters should be debrided as they may contain sequestered chemicals. If blister fluid becomes purulent or the blister leaks, then it should be debrided. Any loose skin or eschar should be mechanically debrided to

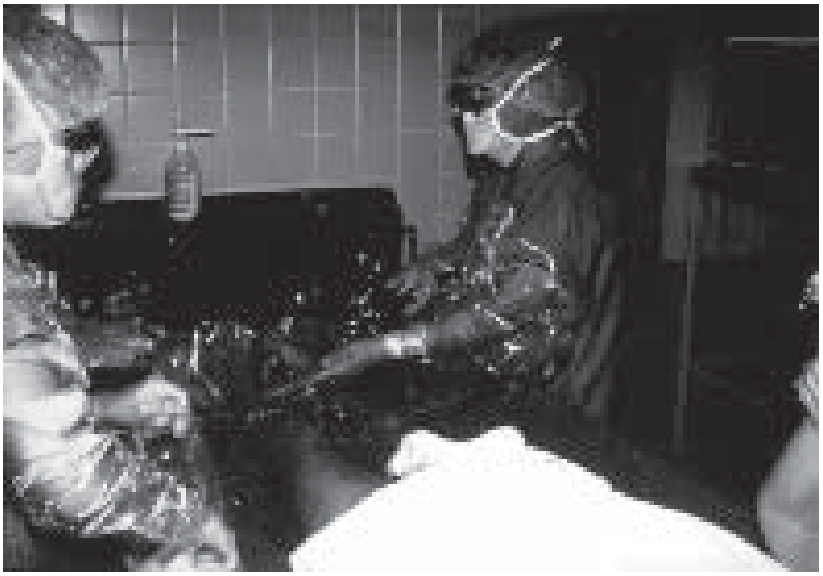


Fig. 10-26. Shower trolley method of wound cleansing. Note the protective barrier clothing worn by the staff.

prevent a breeding ground for infection. Hair harbors bacteria and should be shaved in the burn wound and clipped or shaved around the burn wound.[52] The eyebrows are an exception and should never be shaved.[54] As hair bearing areas heal, hair follicles may remain sources of infection and shaving is continued until the wound is closed. After this, dry scalp is cleansed daily. Weekly wound cultures should be obtained to determine the microorganisms present. If the wound appearance or type of drainage changes radically, cultures should be obtained more frequently.

Special attention should be given to the perineum. If an indwelling Foley catheter is present, thorough cleansing of the area is important. Catheters should be removed as soon as possible. Thorough cleansing after each stool is important to decrease wound contamination from stool. If diarrhea is problematic, use of a rectal tube may be considered to decrease frequent soiling of the wounds. The balloon on the rectal tube should be deflated every 2 to 4 hours for 10 to 15 minutes to prevent pressure areas.

The nurse or emergency medical technician at the scene interviews the patient before intubation is done to determine allergies or sensitivities; medications being used; any preexisting disease such as hypertension, diabetes, epilepsy, schizophrenia, or cardiac or renal problems; or other situations such as recent fractures, chemotherapy, and alcohol or street drug use, which might complicate immediate care and rehabilitation.

Exposure to fire, smoke, chemicals, and products of combustion resulting in inhalation injuries can further complicate the burn victim's course of therapy. There may or may not be concomitant cutaneous injuries. A complete respiratory assessment should be based on the history of the injury. Respiratory injury should be suspected if the victim was in a closed space. The length of time the victim was exposed to smoke should be noted and also the types of items burning and the condition of the victim at the scene—burns of the face and neck; soot in mouth, throat, or nose; singed eyelashes, eyebrows, or nasal hairs; tachypnea, cough, hoarseness, or stridor. Diagnostic studies, such as arterial blood gases, carbon monoxide levels, and chest radiographs will provide a baseline to gauge future changes. Because of its high sensitivity and specificity, diagnostic bronchoscopy has become the preferred diagnostic study for identifying inhalation injury.[53]

Airway management in the respiratory compromised patient can be achieved by either endotracheal intubation or tracheostomy. The patient should be allowed to talk to family or communicate with the medical staff prior to intubation if the situation is not an emergency. Nasotracheal intubation is the preferred approach as it is a nonoperative procedure, the tube can be easily removed, and it poses less risk of bacterial contamination of the lungs. The use of a high volume-low pressure cuff is advocated with the cuff pressure adjusted to a minimal leak. Minimal leak technique is done to determine the least amount of air needed to seal the cuff on the trachea. This technique will decrease problems of stenosis and vocal cord damage, fistula formation, tracheal dilation, and allow proper mechanical ventilation. Securing nasotracheal tubes in the patient with facial burns can be achieved by using umbilical tape or twill tape tied securely around the tube, then around the head. As facial edema increases and decreases, adjustments in the ties should be made. Ties should be changed to prevent accumulation of exudate or crusting of the ties.

If orotracheal intubation is used, a bite block should be placed between the teeth to prevent the patient from biting the tube and obstructing it. Advantages and disadvantages are similar to those for nasotracheal intubation plus the disadvantage of decreased ease of providing good oral hygiene in the orotracheal intubated patient.

Tracheostomy may allow for patient comfort and easier oral alimentation. Its disadvantages include increased risk of bacterial contamination of the lungs, trauma to the neck if burns are present, and greater risk of tracheal injury as tracheostomy cuff pressures are higher than endotracheal tubes.[54]

Mechanical ventilation is used in patients not able to maintain adequate spontaneous respirations. There are several respirator modes from which to choose to provide the appropriate therapeutic setting.

Control mode. The patient receives a breath from the ventilator at predetermined rates, whether or not the patient attempts to breathe.

Assist mode (assist/control). The patient receives a ventilator breath whenever an attempt to breathe is made; a minimal rate is provided if no attempt to breathe is made.

Intermittent mandatory ventilation mode. Regular predetermined breaths are delivered and the patient is allowed to breathe at his own tidal volume and, later, between ventilated breaths. This mode is frequently used for weaning.

Positive end-expiratory pressure (PEEP). PEEP improves oxygenation by improving ventilation to poorly or nonventilated lung segments and prevents alveolar collapse.

Continuous positive airway pressure (CPAP). CPAP restores the glottic mechanism of intrapulmonary pressure maintenance, which has been eliminated by intubation.

The use of medications may aid in the management of the ventilated patient. Appropriate analgesics and sedatives will reduce anxiety and allow patient and ventilator to work together. Sometimes the use of muscle relaxants is necessary to assist in this process. In a few cases, paralyzing medications are needed to allow adequate ventilation. Steroid therapy in inhalation injuries remains controversial. Generally, prophylactic antibiotics are not used as this may lead to the development of resistant strains of organisms.

Circumferential thoracic burns may restrict respiratory excursion and escharotomies should be performed to improved respiratory excursion.

Nursing assessments and duties include (*a*) breath sound assessment; (*b*) pulmonary toilet using aseptic suctioning technique; (*c*) notation of color, quality, and quantity of sputum; (*d*) turning patient and positioning; (*e*) postural drainage and chest physiotherapy if tolerated; and (*f*) monitoring for signs of infection and changes in sputum or growth of pathogens, fever, changes in vital signs, or chest pain. They also assist in monitoring arterial blood gas (ABG), chest radiographs, and patient's progress with weaning.

Once the patient is weaned and extubated, aggressive pulmonary hygiene is crucial in maintaining the patient's airway and maintaining pulmonary function within normal limits. Proper care includes frequent deep breathing, coughing, position changes, use of incentive spirometer, or nebulizer treatment, and provision of humidified oxygen. Suctioning may be required if the patient is not able to adequately clear secretions. Continued monitoring of ABGs or oximetry and serial chest radiographs is required.[55]

Constipation is a common side effect of analgesics. The nurse must monitor and treat this problem. Stool softeners, bulking agents, or laxatives should be used. Diarrhea may be a sign of impaction, *Clostridium difficile* infection, high osmolarity or high fat tube feeding, and proper evaluation must precede management decisions.

Weekly photographs starting at admission should be taken to document the course of wound healing. Photographs should include overall body areas as well as close-ups of individual areas and should be taken after cleansing and before dressing applications. Close-up photos with a measuring device next to the small open area or a decubitus are useful.

The nursing staff should closely examine splinted areas during dressing changes to detect pressure areas. Occupational therapists (OTs) and physical therapists (PTs) should periodically see the casualty during dressing changes. During this time, they can monitor range-of-motion without restrictive dressings, view open areas or areas of potential problems, and formulate a plan of action.

Awareness of environmental room temperature is important so the burn casualty undergoing wound cleansing does not become hypothermic or waste calories maintaining body temperature.

Room temperatures in the 80°F range are generally comfortable for the casualty. This temperature can be maintained by thermostat adjustment to increase the individual room temperature or by directing heat energy to the casualty using overhead heat lamps or heat shields.

The nursing staff monitors the casualty's level of pain and anxiety throughout the day as well as before and during painful procedures. Adequate analgesics are administered before and throughout these procedures. Patient controlled analgesia (PCA) is often appropriate when the casualty has an intravenous (IV) infusion in place. Long acting narcotics such as continuous morphine or methadone are given to relieve background pain that is continuous when other painful treatments are not occurring. Other relaxation modalities, such as deep breathing, guided imagery, and listening to music can also be instituted by the nursing staff to provide distraction and emotional support. These relaxation techniques should be introduced to the casualty and practiced by the casualty prior to painful procedures. Some casualties find they can also control their pain by participating in their care, whether it is helping to remove dressings or assisting in gentle wound cleansing. When casualties perceive they have some control over their care and treatment, anxiety, apprehension, and often pain, decrease.[56]

Burn wounds can be treated using an open or closed technique. There are advantages and disadvantages to both techniques. In the open method the topical antibacterial cream is applied directly to the wound and the wound is left open. This method allows for increased visualization of the wound, mobility unencumbered from bandages, requires less initial nursing time, and reduces cost of bandages. Disadvantages to this method may include increased risk to the casualty for hypothermia and trauma to the wound. Nursing staff must frequently reapply the topical cream as it dries out or is wiped off. Also, the sight of the open wound may be difficult for the casualty, family, and visitors.

The closed method utilizes a variety of gauze and or specialty dressings commonly used on burn wounds. This method of dressing usually consists of three layers.

1. Contact layer, such as fine mesh gauze or Adaptic (Johnson & Johnson), that transports secretions from the draining burn or acts as a protective barrier for healing wounds. This layer can be impregnated with the antibacterial cream.
2. An intermediate layer, such as coarse mesh gauze or burn pads, which cushions the wound and absorbs drainage.
3. The outer layer, consisting of Kerlix (Kendall Healthcare Products Co.) or Kling (Johnson & Johnson), which helps keep the other layers in position. It should conform to the body part in such a way as to avoid constriction secondary to edema.[52] Products such as Burn Net (ACME) also aid in keeping dressings in place without using tape. Special attention should be paid to fingers, toes, or areas where burn surfaces are adjacent. These areas should be individually wrapped so burn surfaces do not touch each other. This will help prevent mechanical trauma and minimize wound contractures.[52] Gauze should be wrapped distal to proximal in a gradient manner to allow for better circulatory return.

Nursing protocols should include proper positioning to prevent contractures and reduce edema. Extremities should be elevated above the level of the heart to decrease edema formation, promote venous return, and decrease pain. Frequent checks of circulation and peripheral pulse are important to detect circulatory compromise. Casualties with burns of face, neck, and head should have the head of the bed elevated to assist edema control and respiration. Also, casualties with head and neck burns should not have pillows conducive to flexion of the neck. Pressure on ear burns can be relieved by using plastic protectors, or foam donuts placed under the head instead of pillows. Care with tie tape placement for airways and nasogastric tubes must be taken to decrease the risk of chondritis. If the patient develops sinusitis from nasal airways and nasogastric tubes and these tubes cannot be discontinued, they are either changed to the oral types or a tracheostomy is performed. There is no definite time for a tracheostomy; it is left to the discretion of the surgeon in consultation with the pulmonary team. If the patient is going to require an airway for pulmonary toilet or ventilator use for a prolonged period of time a tracheostomy is preferable. Sometimes a tracheostomy is used to protect new fragile face or neck grafts.

Nurses monitor and apply the orthotics provided by the therapy team to ensure 24-hour compliance with proper alignment without decubiti. It is important for the primary nurse to facilitate open communication with the burn team members in regard to scheduling the casualty's activities to provide time for medical treatments, occupational and physical therapy, and rest periods. Involving the casualty in the scheduling process frequently reduces casualty stress and promotes acceptance. Posting the casualty's schedule and any special routines regarding splints or exercises will also increase casualty, family, and staff agreement when an OT or PT is not working with the casualty. Optimal outcomes are achieved more readily with team consistency and harmony.

Ambulation or sitting up in a chair the day after injury, if not contraindicated, assists improved respiration; prevents pneumonia and orthostatic hypotension; and also assists the casualty in regaining strength, maintaining mobility, and feeling less passive. Casualties with leg burns should wear single or double elastic wraps when the legs are in a dependent position to increase venous blood return and decrease the pain associated with venous stasis (Figure 10-27). Cotton batting padding strips may also be needed on either side of the tibia to protect thin tissue over this bony prominence in the very old or thin person.

Whenever possible, functional activities around the clock should be encouraged. Not only should the burn team be aware of what the casualty is capable of doing in areas of self-care, but the family should know as well. Frequently, the burn team and family will do activities for the casualty that he is capable of independently performing. The casualty gains self-esteem and self-confidence by being as independent as possible. The family can be directed to participate with the casualty in activities such as applying lotion, organizing get well cards, answering mail, or playing games, rather than promoting casualty dependence by feeding the person.

Casualty and family education is an important factor in understanding and executing the treatment program. The nursing staff must be able to assess the casualty and family's readiness and ability to learn. Apprehension can be decreased and motivation increased with careful explanations and rationale given for all procedures. When the family is included, and has a good understanding of the burn treatment program, they are able to give better support to the casualty. Family members and the pa-

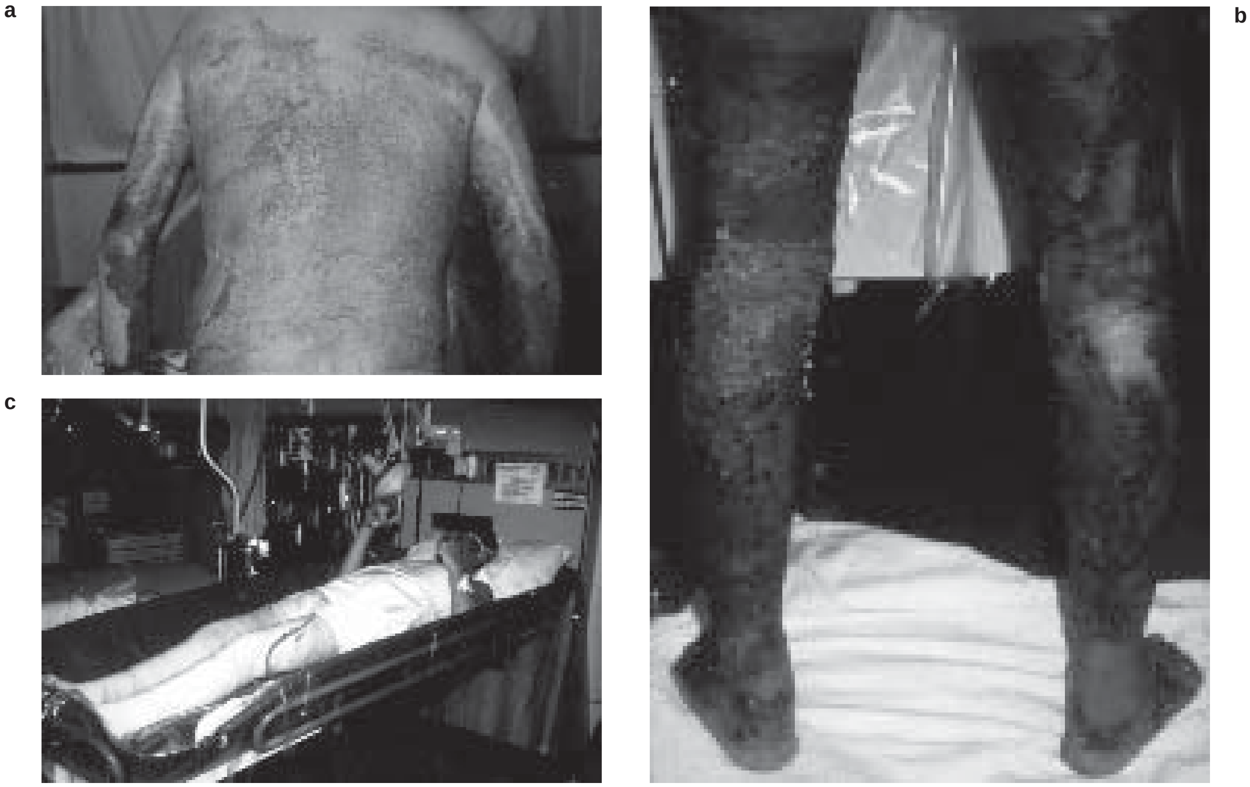


Fig. 10-27. Venous stasis, purple color of dependent tissue and proper elastic wrap support. (**a**) Venous stasis in dependent areas. (**b**) Blisters and vascular insufficiency in legs without venous support. (**c**) Double elastic wraps applied prior to dependent positioning or ambulation.

tient supply important information about the individual's past and present thoughts, feelings, wants, needs, and learning style, which contribute to more effective rehabilitation.

For the burn casualty, adequate nutrition is as vital as any other phase of wound management, for without it, wounds, grafts, and donor sites will not heal and the casualty becomes more susceptible to infection. The nutritional goal in the burn casualty is to achieve a positive or neutral nitrogen balance. Patients with minor burns under 20% TBSA usually can meet their needs with a high calorie, high protein diet and supplemental multivitamins. Casualties with more than 20% TBSA burn may require nutritional support via a feeding tube. Frequently, paralytic ileus is a complication found in the major burn casualty. The ileus generally will resolve in one or two days. With the restoration of bowel sounds, enteral feedings of an appropriate tube feeding supplement can be initiated.[48] These tube feedings can be infused continuously or intermittently by a controlled pump. Depending on the type of tube feeding product used and casualty tolerance, initial administration of hypertonic tube feeding, such as Traumacal (Mead Johnson) should be half strength at an infusion rate of 50 cm^3/h for the first 24 hours, then gradually increasing the rate and then the strength over the next few days. If continuous enteral feedings are used, it is important to check stomach residuals every four hours to ensure the stomach is emptying and prevent aspiration. This can be done by using a piston syringe, aspirating back the stomach contents, recording the amount, and returning the aspirate to the stomach. If the aspirate is greater in mL than 110% of the hourly rate or if the casualty complains of a very full feeling, the tube feeding rate should be decreased or tube feedings held until the residual decreases. Feeding tubes can be held in place by conventional taping methods provided the burn does not involve the face. If facial burns have been sustained, twill tape can be secured around the tube and then tied around the casualty's head (Figure 10-28). The knot should be on the side so the casualty does not lie on it. Also, the ties should be snug but not too tight and should be closely evaluated as edema fluctuates. Twill tape ties should be changed when face care is done.

Daily weights and calorie counts are important in assessing the casualty's nutritional intake and in evaluating progress. As wound size decreases and metabolic demands decrease, the caloric and protein requirements should also be reassessed to avoid overfeeding and excessive weight gain.[48] As the casualty becomes able to tolerate adequate oral nutrition, the enteral feedings are decreased. When nausea or vomiting are problematic, the nurse assesses the cause and, after consultation with the physician, alters the feeding program or administers antiemetics. Casualties with persistent ileus or other complications that would prohibit the use of enteral feedings will require parenteral hyperalimentation and IV fat to meet their nutritional requirements.[48]

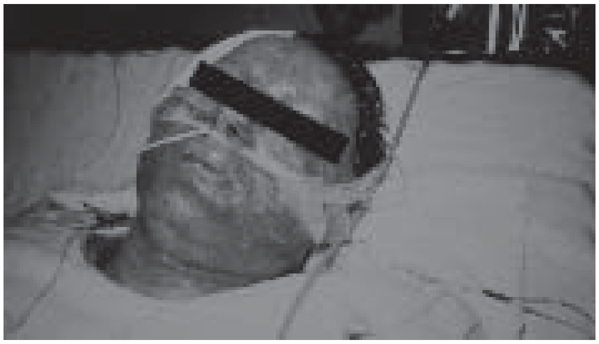


Fig. 10-28. Feeding tube secured with twill tape.

Exercise during the acute phase of healing. For the purpose of discussion in this chapter, the types of range-of-motion will be defined as follows:

- Passive range-of-motion (PROM) is movement of the joint through the unrestricted range of motion, which is produced entirely by an external force and there is no voluntary muscle contraction. It is pain free, resistance free and is short of the anatomical end points of the joint or of the limit if a contracture or calcification are present. Movement performed without assistance or resistance on the part of the patient is considered passive exercise.
- Active assistive range-of-motion (AAROM) is movement in which assistance is provided by an outside force. The prime mover muscle for that joint needs assistance to complete the unrestricted motion. AROM with terminal stretch means the patient vigorously moves the joint through available motion, then the therapist gently stretches the joint in its proper plane of motion, toward the extremes of full motion. This is often used for burn patients to elongate contracting connective tissue in the healing wound. During the stretch the reddened,

healed wound blanches and then turns pink again. AROM is movement within the unrestricted range of movement, which is produced by active contraction of the muscles crossing that joint.[57]

- Prolonged stretch is a relatively long term position or equipment controlled stretch, characterized by objective torque application. It is different from all other range-of-motion definitions.
- Low load is applied for a very long, tolerable time relative to the load the joint is able to take. This type of stretch is used overnight or for a prolonged time and therefore must be tolerable for the long term benefit. It is similar to serial casting. Natural healing of burn wounds occurs by contraction, and this shortened connective tissue is particularly responsive to this sustained type of stretch. Low load, prolonged stretch has the additional benefit of gently, slowly overcoming muscular cocontraction by the casualty who is fearful of pain or who is confused and resisting all exercises. Casualties often request desensitization by vibration or massage of adjacent areas to assist them to relax during this type of stretching. Massage directly on the scar is often painful and until the healed scar is durable, may produce painful blisters, so is contraindicated. Distraction of joint surfaces in conjunction with this stretching may decrease pain and muscle spasm and increase the effectiveness of stretching.[57]

A thorough history is needed to determine the type of range-of-motion that will meet the casualty's, physician's, and therapist's mutual goals. Passive stretching is never used in the case of proximal interphalangeal (PIP) joints that have exposed tendon or joint capsule or burned elbows. Active motion decreases the risk of heterotopic ossification at the elbow joint.[58,59] All range-of-motion is contraindicated in cases of torn ligaments, tendons, or muscles; in the region of unhealed fractures; and immediately following surgical procedures to the skin, tendons, or surrounding soft tissue. Soldiers who sustain battlefront burns often sustain accompanying injuries such as penetrating wounds, fractures, or concussions in addition to skin damage.

Muscle soreness can be avoided by light warm up and warm down activities. When severe joint or muscle pain is noted during exercise or when pain lasts more than 24 hours, the therapist should evaluate the cause and change resistance, duration, speed, and frequency of exercise appropriately. When pain persists, consultation with the referring physician is also indicated.[60] Burn casualties seeking pain relief are often seen lying in a flexed, adducted position. This is the position of withdrawal from pain. When contractures develop, they are most commonly noted over flexor surfaces. Contractures in a flexed position are very difficult to alleviate because extensor musculature is usually much weaker than flexor muscles. An extended position is equally comfortable as flexion, once the tissue stretches. Immobility or rest does not relieve pain as well as analgesic medications; in fact, immobility increases pain because initially skin and later joint contractures quickly become resistant to being stretched. Casualties, friends, and relatives often believe the myth that "rest is the best way to heal a burn." However, burned soldiers do not become strong and flexible by passive motion or rest.

Therapists are encouragers. The acutely burned soldier, with the assistance of sufficient analgesics and sedatives, must move gently through full AROM each day to improve peripheral circulation, keep joints nourished, and prevent contractures or decubiti. The therapist is the resource. The casualty is the source of exercise and recovery. Together they collaborate to form the rehabilitation plan most appropriate to this individual injury, with foremost emphasis on active motion.

AROM is the only successful method of keeping healing burned tissue elongated. All areas injured, including the face, neck, bilateral upper extremities, hands, bilateral lower extremities, feet, and trunk require attention. Contractures of the flexor surface such as finger PIP, knee, and elbow flexion are noted most frequently. However, exercise in all planes of motion must take place. The core musculature receives primary consideration in order to stabilize the trunk, neck, and scapula during extremity exercise. It is a challenge to initiate active motion as soon as the casualty is admitted, especially when the injury is greater than 50% TBSA and occurred in combat. However, since passive treatments are ineffective in full recovery, it is important to provide analgesia and sedation levels adequate to enlist the casualty in early exercise such as bed mobility, reasonably comfortable dangling, getting up for bathroom care, sitting in the chair, and ambulation. The tilt table is an excellent preparation for ambulation when the casualty is too weak to ambulate independently (Figure 10-29). Tilt table positioning is begun before grafting is completed. Often the soldier will have many IV lines, a Foley catheter, and

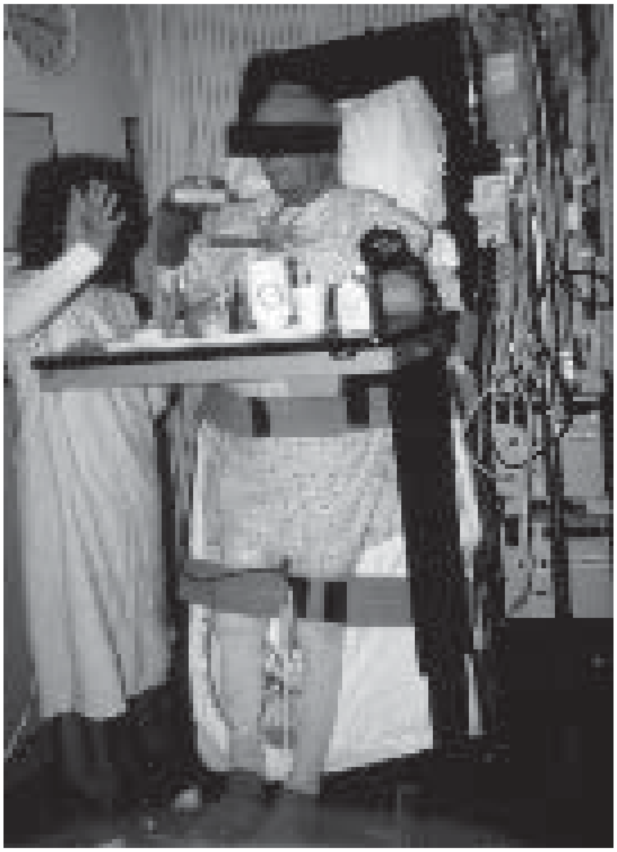


Fig. 10-29. Standing table exercise. Daily living skill practice distracts the casualty during standing.

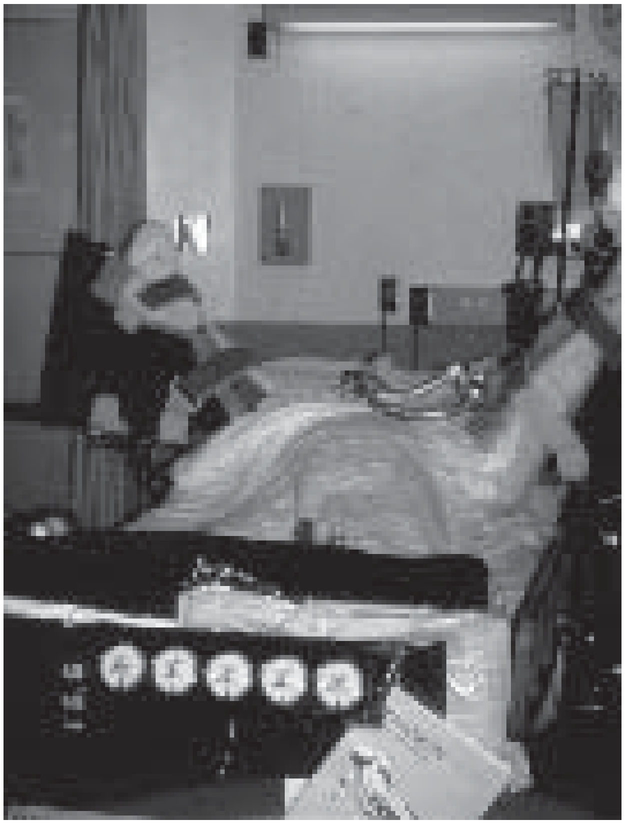


Fig. 10-30. Kinaire bed anticontracture bed positioning, neck extension, shoulders abducted to 90°, arms on wedges, hips and knees straight, and ankles at neutral.

chest and feeding tubes in place when the tilt table exercise is initiated. Tilt table exercise will often be combined with functional activities to help distract the person from wanting to return to bed.

The alert, cooperative casualty must learn to move past the painful range to the extremes of joint motion. Gentle terminal stretching is the least painful method of achieving this mobility. Active assistive exercises with terminal stretching teach the casualty how to move the body part and achieve the extremes of motion which are not used spontaneously during activity (Figure 10-30). Prolonged, vigorous stretch, although sometimes appropriate with a healed, contracted joint, is never appropriate with the early edematous burn. For the receptive casualty, PROM is unnecessary.

Exercise can be done during the bathing procedure and at other times during the day. If the bandages are dry and stuck or the xenograft has dried and is inflexible, pain will inhibit cooperation with full AROM. In this case, exercise during the bathing procedure will benefit the casualty; however, he may be distracted by wound pain, may not be able to do active motion, and pain medications will inhibit his ability to remember the exercises. If there is topical medication under the gauze bandages and they are wrapped in a loose figure-8 method that slides easily, exercise in the bandages is preferred by the casualty. In all cases the therapist and prescribing physician should observe the tissue with bandages removed during the stretching exercises at least twice a week.

Walking is one of the more comfortable early exercises. The use of an overhead walker provides a graded method of elevation and exercise for the upper extremities (Figure 10-31). Additionally, squeezing the overhead bar facilitates a "pumping action," thereby reducing edema in the upper extremities. The overhead bar is large enough to avoid damaging inflamed dorsal finger tendons during exercise. During the early treatment period, ambulation 3 or 4 times a day for 15 minutes, using the overhead walker, will maintain satisfactory shoulder flexion and elbow extension; wrist extension; gross fist of hand; and straight trunk, hip, knee, and ankle flexion and extension. Gentle daily elevated

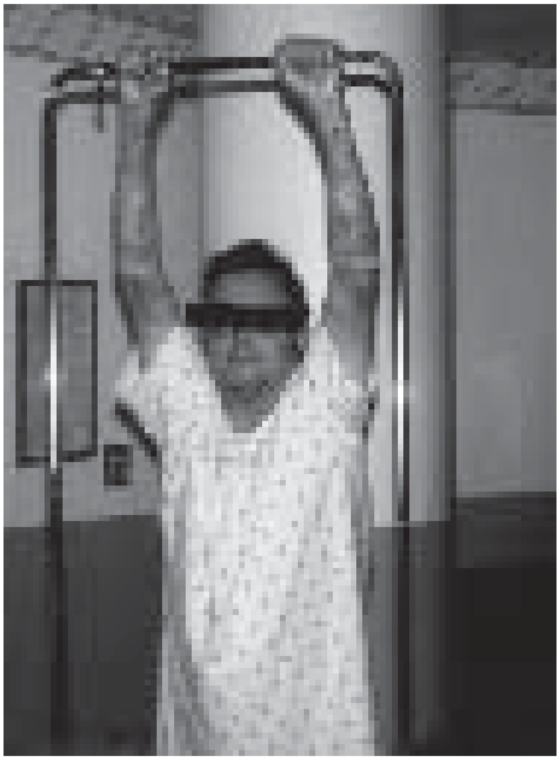


Fig. 10-31. Overhead bar connected to a wheeled walker used for hand exercise, arm positioning, and shoulder flexion.

active exercise not only prevents skin or soft tissue contractures, it also improves circulation and decreases complications such as pneumonia, pulmonary emboli, phlebitis, and venous engorgement. When exercise decreases edema and inflammation, the wound heals more quickly and there is less pain. Physical activity gives a person increased energy, and decreases depression[61] and insomnia, which also speeds healing.

Continuous passive motion (CPM) devices help to modify edema[62] and preserve motion when the joints and soft tissues are not yet stiff. However, casualties who are recovering from larger burns begin to develop stiff joints and contracting soft tissue during the second or third week after injury. Reciprocal pulleys, two-handed calisthenics, slow bicycling, and dowel exercises (Figure 10-32) have been safe methods of providing stretching motion. The casualty controls the speed and duration of the passive stretch. The therapist is the coach. Foam or rubber band grippers are safe early hand exercise devices. Liberal exercise choices and written graded programs assist when a casualty is hostile or obstreperous. It is reassuring to the casualty to be given as much control as possible in exercising, since he does not have control of most other treatment areas.

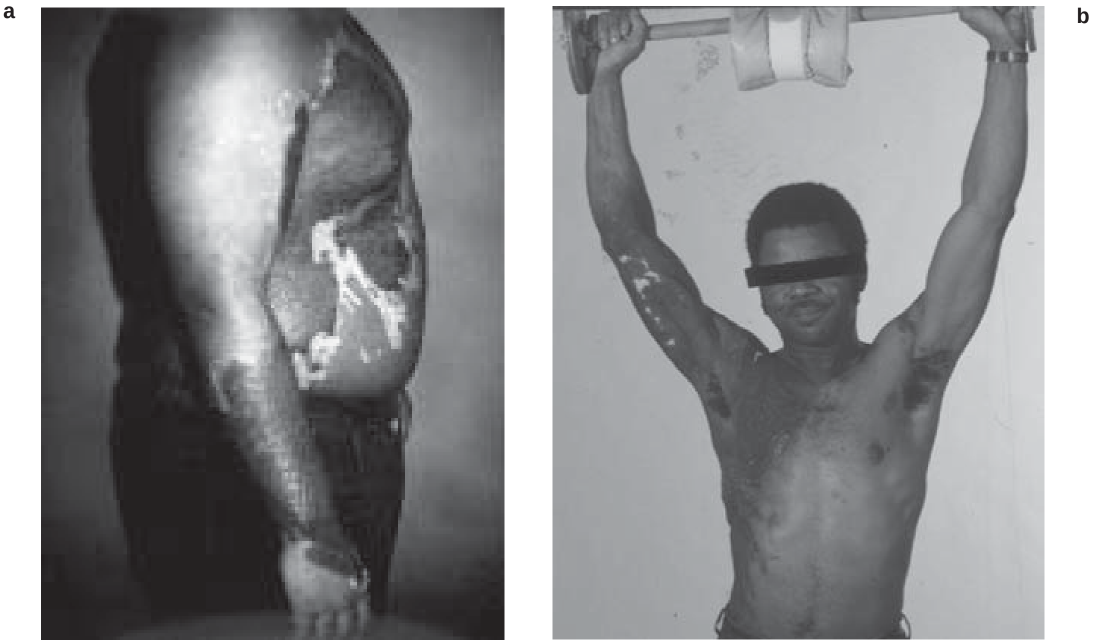


Fig. 10-32. Pigmentation of mature burn wound. (**a**) Hypopigmentation and hyperpigmentation, Caucasian person. (**b**) Hypopigmentation and hyperpigmentation of right arm and chest, African American person. Note the white areas on upper arm represent total permanent loss of pigmentation. Note the patient is using a dowel for overhead exercise.

After several weeks, when the casualty is not maintaining full AROM, it is very helpful to evaluate the range during anesthesia for some other purpose such as skin grafting. If full range-of-motion is present, the casualty should be encouraged to increase the frequency and vigor of independent elevated exercise. When inflammation is present, passive motion must be especially cautious. The therapist moves the joint very slowly and steadily in the proper plane of motion. The joint above and below the one being evaluated are stabilized. Gentle force; slow, steady speed; and correct orientation are the keys to safe range-of-motion. This is particularly true for evaluation when the casualty is anesthetized. The arm is not hyperextended or the shoulder stretched above 120° when the patient is under anesthesia.

The contracted elbow joint or the open PIP joint are never stretched passively, even under anesthesia. It may be possible to allow gravity to stretch the elbow joint into flexion or extension, if "gravity assist" positioning is permitted by the surgeon. In the case of serial or "drop out" casting, an elbow joint may be safely returned to the previous position in the cast if the soft tissue has not contracted more than 1 or 2 hours or the cast has not been off for a prolonged time (Figure 10-33).

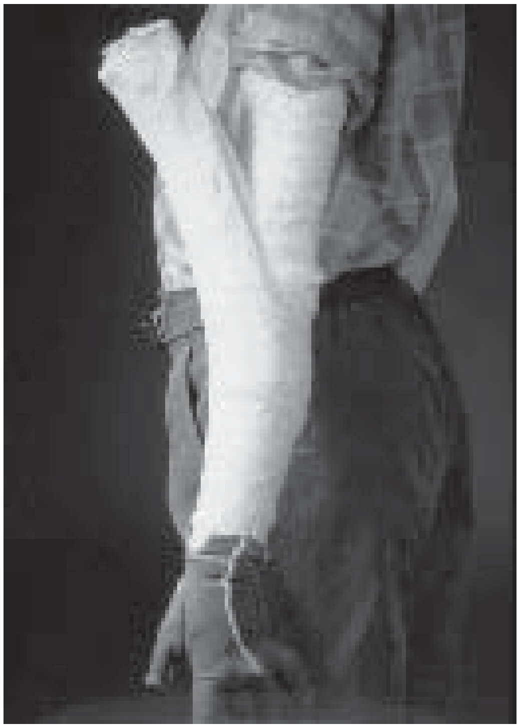


Fig. 10-33. Drop-out elbow orthotic.

When the casualty is too ill for active motion, muscle atrophy and joint contractures develop quickly. Inflammation may contribute to heterotopic ossification or fibrosis.[63] Passive motion in the correct planes of motion for each affected joint is indicated except for the elbow joint or open PIP joints. Passive motion helps to prevent contractures, but will not prevent muscle atrophy. When the casualty is confused and combative, passive motion is indicated. However, when the casualty is causing increased inflammation by fighting restraints, positioning appliances, or passive motion, increased heterotopic ossification, wound breakdown and nerve damage are often observed. Team consultation is appropriate to determine safe parameters of increasing analgesia or administration of major or minor tranquilizers.

Electrical neuromuscular. Functional electrical stimulation (FES) has been used in general rehabilitation with the goals of retarding disuse muscle atrophy, reducing contractures due to weak muscles, and increasing venous and lymph flow in the stimulated muscles. It may be technically difficult to perform with burn patients during the acute stage. It does not replace AROM exercise and is not as effective as an active strengthening therapeutic treatment program even when used at a high current intensity. FES may not be comfortable or well tolerated on unhealed burned tissue. Its use in burn casualties may be better reserved for the maturation phase of burn care.

Intermittent compression using elastic wraps can be very effective in edema reduction when coupled with elevated positioning and/or elevated exercise. The elastic wrap needs to be applied uniformly to prevent a tourniquet effect and must have a greater perpendicular pressure distal to proximal. A mechanical device such as a Jobst Intermittent Compression Pump or a Wright Linear Pump (Wright) can be used when the patient is immobilized or resting. These two devices apply pneumatic pressure onto an extremity at selected pressure and time intervals. The pressure and time must be individualized to be within a patient's tolerance, but the pressure and time should be increased for maximum effectiveness. The pressure should not exceed the diastolic blood pressure. A typical Jobst pump starting pressure may be 40 mm Hg with a time interval of 1 minute "on" and 30 seconds "off" with the "on" time increasing to tolerance. The Jobst pump pressure is uniform throughout while the Wright pump delivers a sequential pressure pattern moving from proximal to distal allowing a "pumping" action to the edematous tissue. A typical beginning Wright pump program would be distal chamber at 45 mm Hg, middle chamber at 25 mm Hg, and proximal chamber at 5 mm Hg pressure; a 2-minute cycle with the distal cell inflated for 90 seconds, distal plus middle cells inflated for 70 seconds, and all three cells inflated for 50 seconds, with a 30 second "rest" or "off" period. These devices should be considered when edema resolution can not be accomplished in another, more conservative fashion. They may not be well tolerated, and increased analgesia will probably be required. The overall treatment time using the pump is increased from 1 hour to overnight as tolerated. The pumps are more commonly used in the maturation phase of healing when the skin can better tolerate their use.

Daily, gently graded activity will diminish loss of strength, while it improves range of motion. This is accomplished by every member of the rehabilitation team actively encouraging the casualty and family to participate in self-care activities and continued exercise, despite the seriousness of the injury. The casualty is motivated by the increased speed of healing caused in part by exercise and activity. Presenting numerous different types of activity helps the casualty choose an activity that is interesting. Nerf games, bicycling, stair climbing, balloon boxing, and calisthenics are good exercises. Crafts, if volunteers or family members join the casualty in these projects, will reinforce activity.

Daily living skills such as independent use of the phone, self-feeding, shaving, brushing teeth, and self-toileting are excellent exercises for the upper extremities. Successful independence is motivating. Unlike repetitive calisthenics, which may be boring, a patient understands the importance of daily living skill practice and is usually motivated by a desire to regain independence. It is helpful for the casualty to keep in mind the goals of regaining maximum independence with the fewest possible scar bands and with the least possible disfigurement while he struggles with self-care or does uncomfortable exercises.

Positioning in the Acute Phase

Proper positioning is the alignment of body parts that is recommended for the burn casualty who is at rest, sitting, lying prone or supine, or standing. During the acute phase, antigravity positioning decreases the potential to develop contractures; assists venous return, which will minimize edema; protects the peripheral nerves from being stretched or further traumatized; assists proper respiratory function; and protects the healing wound. A past medical history of previous arthritis, strokes, brain injury, or residuals from previous trauma may modify the positioning plan. A thorough physical examination and sensory evaluation reveal accompanying injuries that must be considered when developing plans for prolonged positioning. After a burn injury, the inflammatory response initiates a process by which any position maintained for more than 8 hours without active motion causes early contracture formation.

The typical anticontracture bed positioning method would consist of neck extension, shoulders abducted to 90° and forward flexed 15°, elbows lacking 15° of extension and supinated, wrists and hands in functional position, hips extended and abducted 10° without external rotation, knees in extension, and ankles at neutral. There is, however, no single position that totally prevents contractures. (Typical anticontracture positioning—supine, prone, lateral decubitus are shown in Figures 10-30 and 10-34 and are detailed in Table 10-3.)

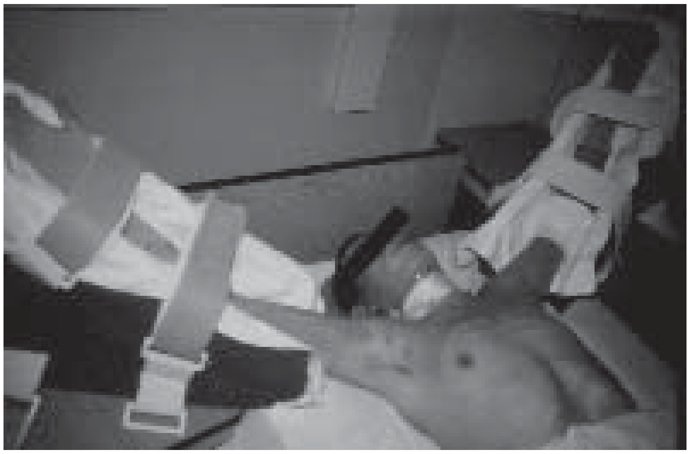


Fig. 10-34. Regular bed with hyperextension mattress anticontracture positioning, head on foam donut, arms on wedges secured to over-the-bed tables.

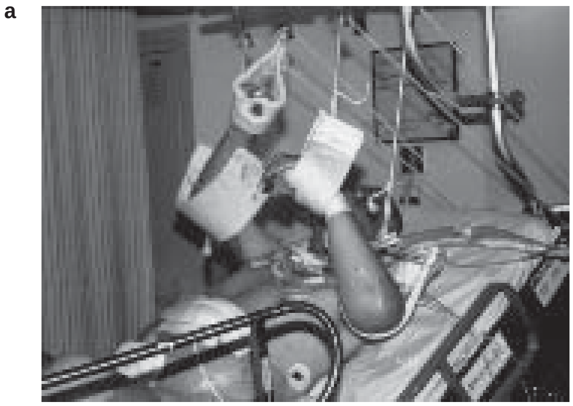
TABLE 10-3

TYPICAL ANTICONTRACTURE POSITIONING—SUPINE, PRONE, AND SIDE-LYING

	SUPINE	PRONE	SIDE-LYING
Anterior neck burns	Position in extension or hyperextension. A roll may be placed under the neck. A towel roll under the shoulders or along the spine hyperextends the neck. If contracture develops apply a soft neck collar.	Alternate head to side.	Position the neck in extension.
Shoulder burns	At 90° of abduction and 15° forward flexion (airplane position).	Abducted and externally rotated as possible.	Alternately position shoulder in 90° of flexion, elevate the arm above the level of the heart.
Wrist/Hand	Elevate wrist above elbow and elbow above shoulder. If contractures develop, apply antideformity burn wrist/hand orthoses.	If no orthosis, use glove vascular support for edema control.	If no orthosis, elevate the free wrist/hand.
Hip	Place hips in neutral rotation, 15° abduction, and extension.	Place hips in neutral rotation, 15° abduction, and extension. Avoid frog leg position.	Alternate right and left legs into flexion and extension.
Knee	Position in extension.	Position in extension.	Position the free (top) leg in knee flexion and the other leg in knee extension. Alternate.
Ankle/Foot	Elevate for edema control. Use AFO to control inversion/eversion. Position ankles in 90° of dorsiflexion.	Position ankles in 90° of dorsiflexion.	Position ankles in 90° of dorsiflexion with AFO. Elevate the free leg with a pillow to prevent pressure over the malleolus. If possible, alternate sides frequently.
Anterior elbow burns	Position in 15° flexion. If flexion contracture develops, position in extension using anterior orthosis. Midposition of supination/pronation.	Position elbows in extension when shoulders are not externally rotated.	Position upper arm in extension.

Each burned part of the body must be considered when planning positioning.

1. Ears: Pillows are removed. A foam donut or bandage positioning is used to prevent ear contact with the bed, (see Figure 10-34) pillows or tie tapes (see Figure 10-28). Irritation of ears increases the risk of chondritis. In the Kinaire (Kinetic Concepts) bed, horseshoe cutout pillows protect the ears and allow neck hyperextension simultaneously.



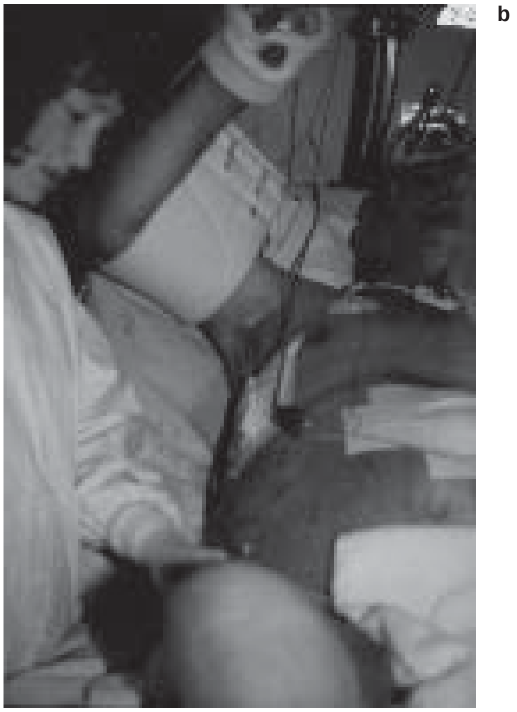


Fig. 10-35. Shoulder positioning in bed, using felt slings. (**a**) Casualty placed at 90° to 120° of shoulder abduction and 15° forward rotation, using felt pressure-distributing slings connected to weighted pulleys and over-the-bed suspension. Right shoulder at 90° abduction, and patient moving left arm toward hip. (**b**) Nurse repositioning patient to reduce hip flexion. This will increase composite stretch while the right axillary contracture web is being stretched with over-the-bed traction .

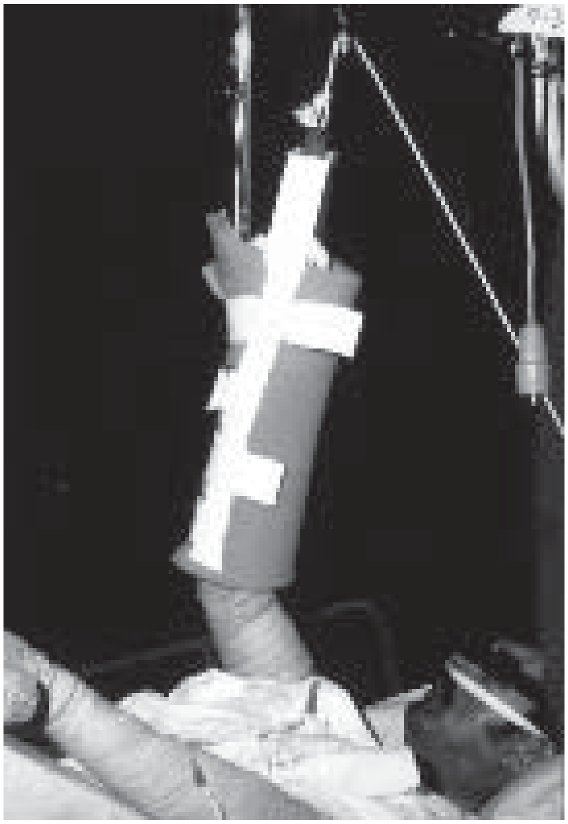


Fig. 10-36. Skin foam extremity suspension assembly. Note that the left hand is elevated on a wedge and MP flexion is preserved with elastic roll in palm.

2. Nose: A foam face support with a cutout should be used when the person is prone. Wide tie tapes are used to widely distribute pressure under the nose when nasal airways or feeding or gastrostomy tubes are being secured. These tubes may become a source for contamination and sinus infections and therefore should be removed as soon as practical.
3. Mouth: When the person is on a ventilator, the bite block safely positions the jaw and oral opening.
4. Neck:
   - Bed: Neck extension may occur over a crescent cutout in the air flow cushion bed, a foam wedge cutout in the fluidized air flow bed, or a foam elevation wedge or short mattress with a regular bed (see Figures 10-30 and 10-34).
   - Sitting: A pillow behind the scapular area will allow neck extension in a recliner chair.
5. Shoulder:
   - Bed: Slings to a weighted pulley and over-the-bed suspension traction support the shoulder at 90° to 120° of abduction and 15° forward rotation (Figure 10-35). An extremity suspension assembly, foam (Figure 10-36), or stockinet suspension to over-the-bed traction positions the arm

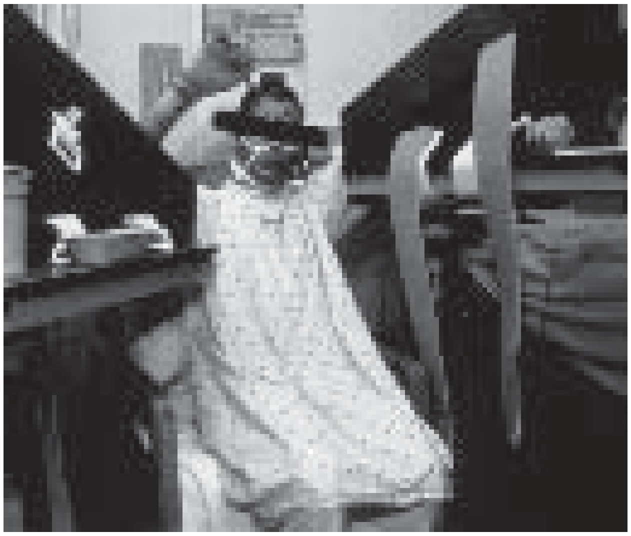


Fig. 10-37. Anticontracture sitting position. Foam wedges are secured to the bedside table.

at 90° forward flexion or a foam arm elevation wedge positions the shoulder in the mid position (see Figure 10-34).

- Sitting: Deltoid aids, foam arm elevation wedges secured to chair armrests or elevating table's position (Figure 10-37) shoulder at 90° and elbow above heart.
- Standing: IV pole or overhead bar connected to a wheeled walker for elevated arm position and shoulder flexion (see Figure 10-31).

6. Elbow: The wedge positions the elbow in supination and lacking 15° of extension to avoid tension on the brachioradialis muscle. It is useful for sitting or supine positions. An IV pole, traction-type bar, or over-the-bed bar is needed for standing.
7. Wrist: The wrist is kept in a neutral position if the hand is strapped into the elevation wedge. If a bandage roll is placed into the palm, the wrist is extended about 15°. In bulky "Robert Jones dressings" for over-the-bed suspension, the wrist is positioned in the mid position. Care must be used to allow Doppler monitoring of peripheral pulses.
8. Hand: When the hand is the only burned area, pillow elevation may be adequate. An arm elevation wedge (see Figures 10-30, 10-34, and 10-37) with an elastic wrap roll can preserve the first web space and decrease edema while protecting the elbow and shoulder (see Figure 10-36). In the acute phase, the fingers should be included in a bulky wrap to prevent impaired distal circulation. If bulky wrapping is not used, the fingers should be wrapped separately to encourage motion (Figure 10-38) and the arm should be elevated.
9. Hip:
    - Bed: The hip is best positioned in full extension with 10° abduction and no external rotation (see Figures 10-30 and 10-34). A trochanter roll may be needed to maintain this position when the patient is supine.
    - Sitting: The hip should be positioned in as much extension as possible if the patient is in a recliner chair, alternating with flexion to 90° unless this is contraindicated because of cellulitis.
    - Standing: The patient should fully extend the hip when walking, and hyperextend the hip with a normal gait pattern.
10. Knee**:**
    - Bed: Knee extension is the position recommended for the majority of time. The "frog leg" position should be avoided to prevent peroneal palsy. When the patient is prone on the air-flow bed, this position is facilitated by tucking the toes between air flow cushions. For short periods of time, the knees may be flexed, as when turning side to side, or just by elevating the foot of the bed.
    - Sitting: The knee should be positioned in full extension, alternating with flexion to

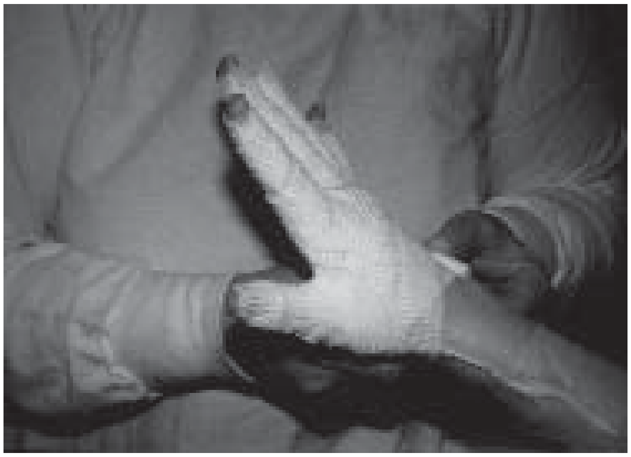


Fig. 10-38. Fingers are wrapped separately in Sof-Kling.

90° unless this is contraindicated because of cellulitis.

11. Feet and ankles:
    - Bed: The Kinaire (Kinetic Concepts) foot cushion is usually adequate unless neurological complications or inadequate analgesia are causing plantar flexion (see Figure 10-30). A cutout in a foam block makes pressure on burned soles of feet more comfortable and can be used for elevation. A foot board can be helpful for ankle positioning when the casualty is supine. Ankle position for prone persons includes placing the foot over the end of the mattress or between foam cushions in Kinaire beds.
    - Sitting: Feet will become edematous if kept dependent for long periods. The ankle can be kept at 90° by the back of a straight chair, if a pillow is placed on the seat to relieve pain and pressure over the calcaneus.
12. Miscellaneous**:** Creative use of slings, sandbags, and sponge positioning devices assist in reducing edema and protecting the healing wound. The ROHO (ROHO, Inc.) bed and wheelchair cushions can also be used creatively for positioning. At times a chair may have the back replaced with a padded dowel to allow air flow to open wounds which are not healing.

Orthosis During the Acute Phase

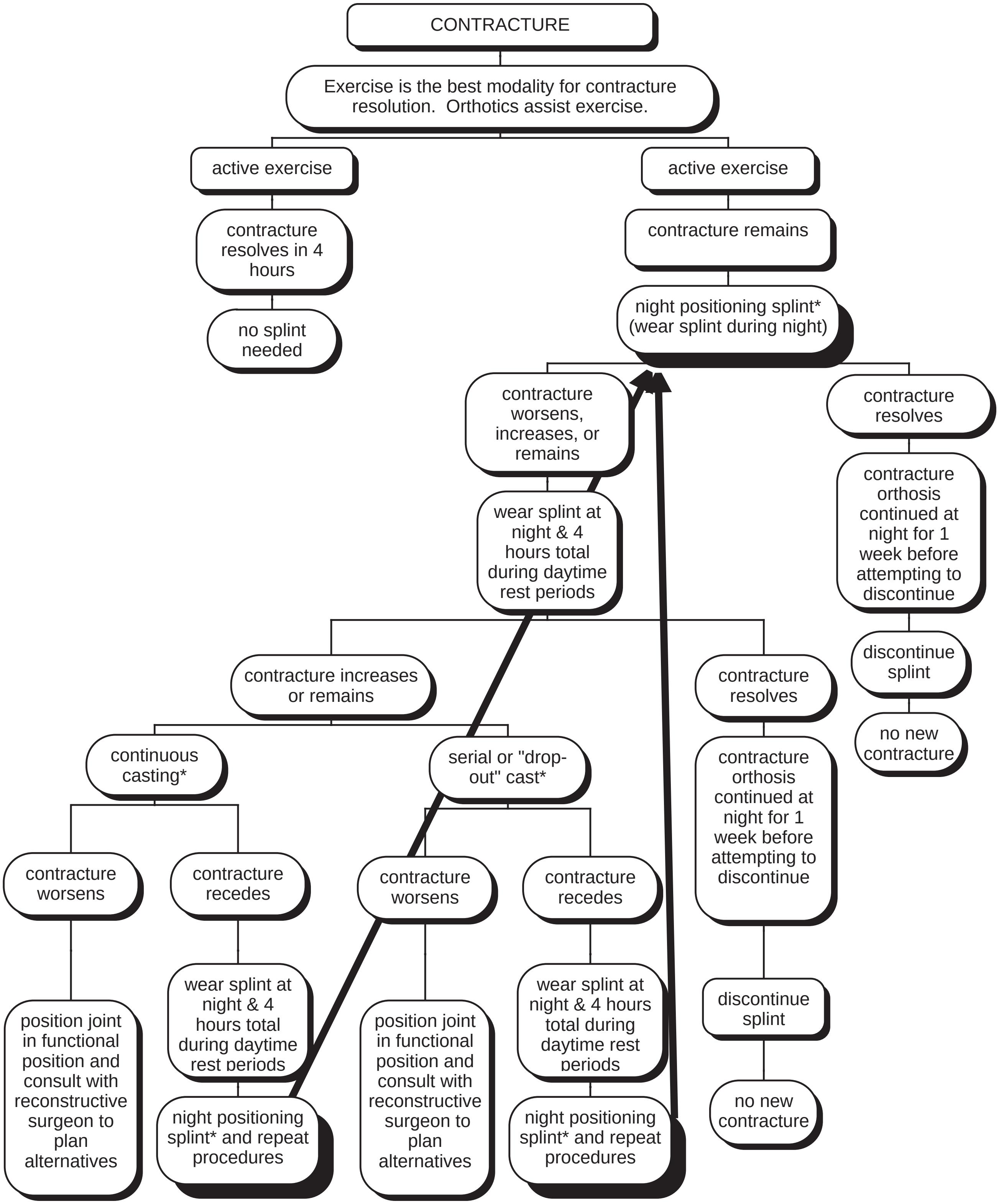
A splint or orthosis is an orthopedic support often used as an adjunct to postburn antigravity positioning. Orthoses are made of low temperature thermoplastic, high temperature plastic, foam, plaster, elastomer or silicone, metal, molded leather, wood, reinforced cloth, and any other material to immobilize a body part or redistribute pressure. An orthosis that has elastic outriggers or attachments to provide stretching or to replace absent function is called a dynamic splint. Static splints are fitted to individual body parts of the burned person who is unconscious or is unable to maintain full AROM by positioning and activity. The purpose of the orthosis in this stage of healing is to immobilize an unstable joint, prevent formation of nonfunctional contractures, keep ligaments in optimal position, protect exposed soft tissue or bone, or assist with antigravity positioning. When antigravity positioning is unsuccessful in managing edema or range of motion, an orthosis is fitted. For the comatose or resistive patient, orthotics may protect the healing wound. Continuous motion splints help reduce edema. However, splints never replace AROM exercises. Resting thermoplastic splints are often custom fitted to the burned person. Sometimes off-the-shelf splints such as LMB (North Coast Medical) foam wire positioning orthoses are prescribed. Whenever possible, active motion replaces orthotics as soon as possible for the optimal outcome.[64]

The indications and contraindications for splinting and casting can be displayed as an algorithm (Figure 10-39).

When evaluating the need for an early hand orthosis, the patient is asked to extend and abduct all fingers and thumb with the wrist in a neutral position. If there is lack of extension of the PIP and distal interphalangeal (DIP) joints (ie, early claw deformity), then a splint may be needed. If this extension lag does not resolve in 4 days, a resting orthosis is definitely needed (Figure 10-40). Often, finger extension troughs or foam extension wraps are used in addition to the resting hand orthosis (Figures 10-41 and 10-42).

The initial burn edema distends the loose, dorsal hand skin, exerting a force on all the joints, which is more exaggerated if the hand is burned. This pulls the metacarpophalangeal (MCP) joints into hyperextension, the thumb into adduction parallel to the second metacarpal, and the inerphalangeal (IP) joints into flexion.[65] The typical resting hand splint is discussed in the next paragraph. When the dorsal hand tendons or joints are exposed, however, the burned fingers are splinted in extension to prevent the central slip of the extensor mechanism from being thinned or stressed. When a rupture of the exposed extensor tendon occurs, the lateral bands of the mechanism slip past the fulcrum of the PIP joint, to become flexors. Over time a boutonniere deformity forms, with the PIP joint in flexion and the DIP joint in hypertension.[66,67] (Figure 10-43) When the extensor mechanism ruptures, the finger PIP joint should be splinted on slack in extension and immobilized until the tendons are granulated, reepithelialized, and stable. This can take up to 6 weeks.[68] After this immobilization, the finger can begin active motion and gradually be stretched into as full flexion as possible. Extension, through the healed scar tissue, may be adequate for most motions.

For a dorsal hand burn a lightweight thermoplastic wrist, hand, finger orthosis (WHFO) is fabricated in the following position[69,70] (see Figure 10-40). Wrist extension of about 20° prevents engorgement in the carpal tunnel area, allows thumb abduction positioning and prevents tenodesis action of MCP hy-


CONTRACTURE
Exercise is the best modality for contracture resolution. Orthotics assist exercise.
active exercise
contracture resolves in 4 hours
no splint needed
active exercise
contracture remains
night positioning splint* (wear splint during night)
contracture worsens, increases, or remains
wear splint at night & 4 hours total during daytime rest periods
contracture resolves
contracture orthosis continued at night for 1 week before attempting to discontinue
discontinue splint
no new contracture
contracture increases or remains
contracture resolves
contracture orthosis continued at night for 1 week before attempting to discontinue
discontinue splint
no new contracture
continuous casting*
contracture worsens
position joint in functional position and consult with reconstructive surgeon to plan alternatives
contracture recedes
wear splint at night & 4 hours total during daytime rest periods
night positioning splint* and repeat procedures
serial or "drop-out" cast*
contracture worsens
position joint in functional position and consult with reconstructive surgeon to plan alternatives
contracture recedes
wear splint at night & 4 hours total during daytime rest periods
night positioning splint* and repeat procedures

•If open area develops, remodel orthosis to relieve pressure.

Fig. 10-39. Algorithm for use of orthotics or casts.

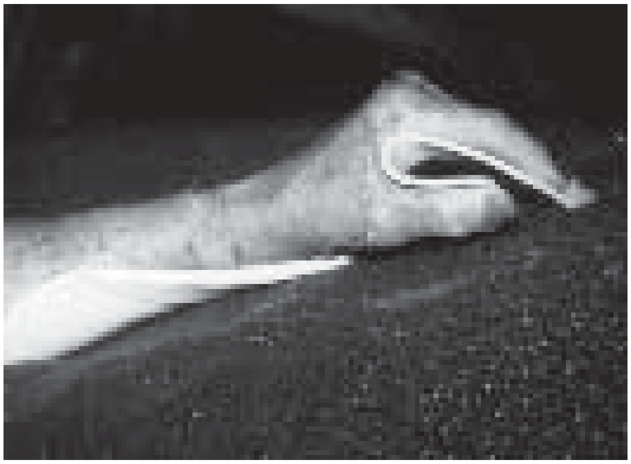


Fig. 10-40. Wrist, hand, finger orthosis for hand. Note that the arm is elevated on a foam wedge with arm cut-out.

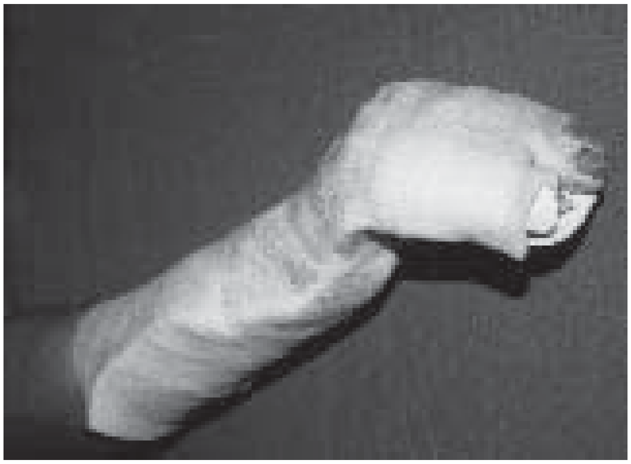


Fig. 10-41. Wrist, hand, and finger orthosis, secured with Kerlix wrap.

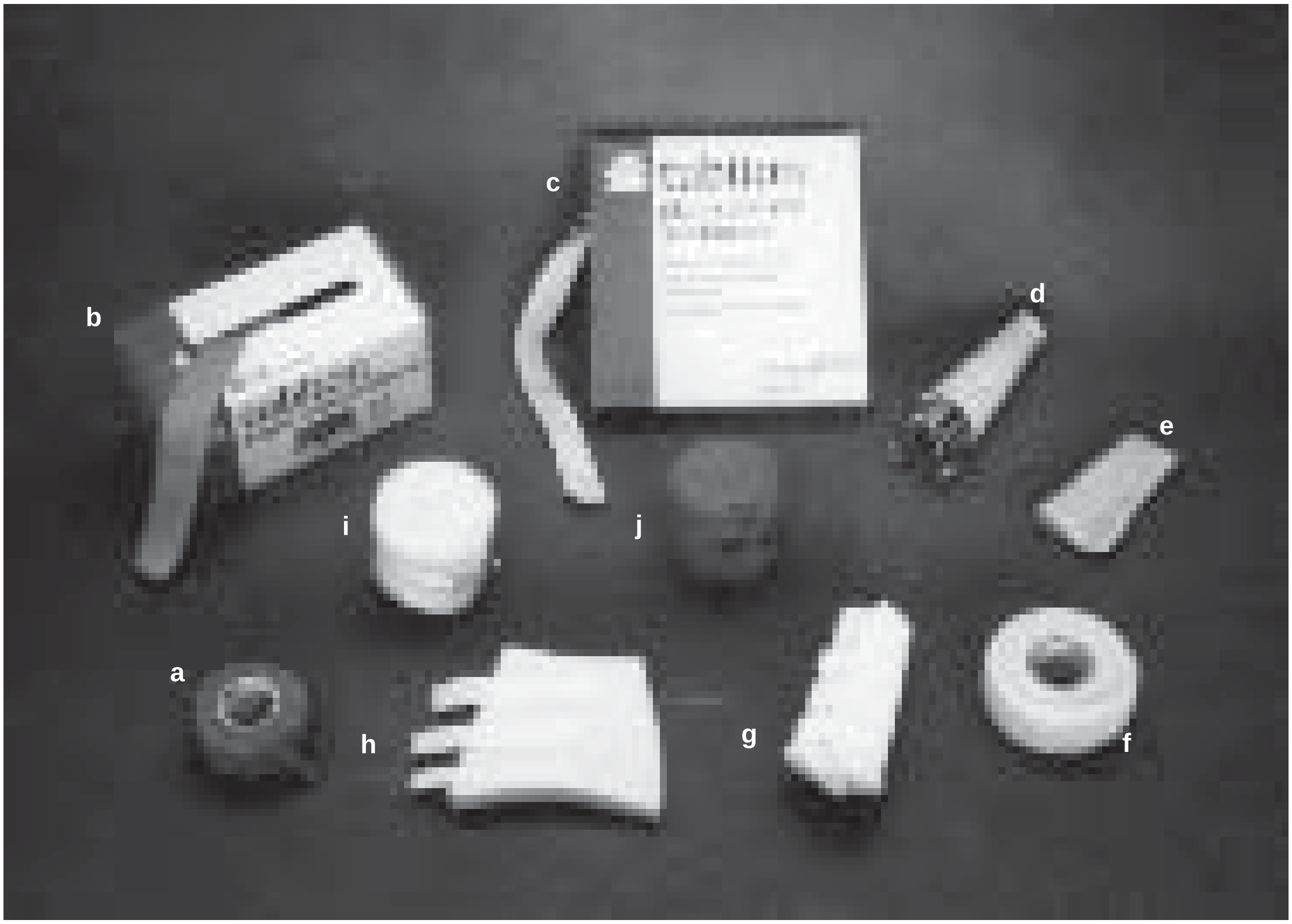


Fig. 10-42. Finger wraps. (**a**) Coban wrap. (**b**) Tubiton Oedema sleeves. (**c**) Regular Tubiton size 01. (**d**) Tubiton on finger applicator. (**e**) Finger elastic wrap sleeve. (**f**) Expandover adhesive wrap. (**g**) Lois M. Barber (LMB) Velcro closure. (**h**) Open LMB finger wrap. (**i**) Else roll. (**j**) 2-inch elastic wrap.

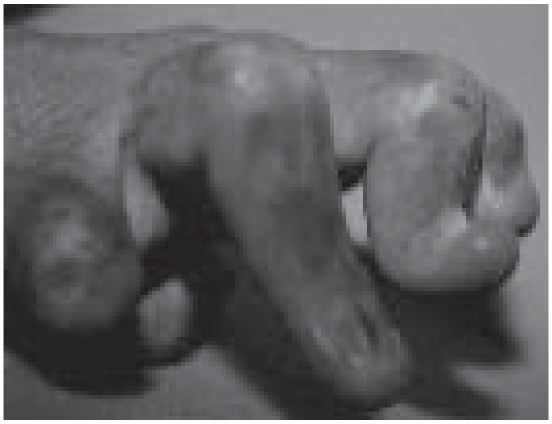


Fig. 10-43. Boutonniere deformity.

perextension, which occurs in wrist drop. Metacarpophalangeal joints are flexed to 50° to 65° with the little finger flexed most, ring finger slightly less, progressing toward the index finger to accommodate the ulnar hand mobility. At 90° or 45° MCP flexion, the cam action of the metacarpal head allows shortening of most of the fibers of the collateral ligaments. In flexion, the cam action also limits most abduction or adduction of the fingers at the MCP joint. MCP flexion, once lost, is very difficult to regain due to complex hand anatomy. Therefore, the MCP joints are positioned at about 55° flexion. The PIP and DIP joints are positioned at 0° flexion, which has been described as the "safe" position or the "intrinsic plus" position. The lateral bands of the interosseous muscles and especially lumbrical muscles (sometimes called oblique retinacular or "Landsmere's ligament"), which originate from the lumbrical and finally attach at the middle and terminal phalanx, are on slack with the PIP joints extended. These may shorten and require slow, prolonged stretching to allow flexing the proximal phalanges when the MCPs are extended. The extensor digitorum longus muscle must be relaxed to allow flexion at the PIP and DIP with extension at the MCP joints. The hand bends at the midpalmar crease not at the junction of the finger with the palm. The splint bend should therefore be seen proximal to the palmar crease. If it is impossible to extended the IPs and flex the MCPs on the palmar support of the orthosis, the splint MCPs are flexed too much given the edema and subsequent loss of mobility. The palmar finger support should not be wider than necessary to support the fingers. If it is made too wide, PIP flexion can more readily occur when the orthosis is secured. Sufficient individual finger wraps at the distal phalanx will prevent lateral finger decubiti. Full IP extension is used for the early splint. Full extension puts collateral ligaments at the PIP joint on stretch. When the burn depth damages the dorsal hood mechanism of the finger and reconstruction is not possible, the hand is positioned in a "functional" position. In this case the functional position that is commonly recommended is 25° flexion of DIP joint and 40° flexion at the PIP joint. This allows functional opposition to the lateral finger to the thumb if MCP motion of the fingers and thumb carpometacarpal motions are preserved. Kept in this functional position, either spontaneous joint fusion will occur if the cartilage has been destroyed by the injury or septic joints or else the fingers will contract to this most functional position. Spontaneous fusion with the joint flexed more than 45° results in a painful and nonfunctional finger. If a mallet finger develops, the DIP joint should be positioned in hyperextension and held there until the extension lag resolves or for 6 weeks to restore the extensor integrity.

The thumb web space is best preserved if the thumb is positioned 15° radially. In addition, the thumb MCP and IP are flexed 5° to avoid hyperextension contractures. The thumb is then positioned away from the palm as far as the hand will allow. This is the most optimal position used to prevent burn deformity (Figure 10-44). This positioning is generally used for all burns, whether circumferential, palmar, or dorsal. If the burn is only palmar, a dorsal, "open palm" splint may be used to immobilize finger tips in extension and maintain a neutral wrist position. This splint must be secured with a wrap that supports circulation in a gradient manner from distal to proximal. Wrist straps are never used. However, when healing is causing a contrac-

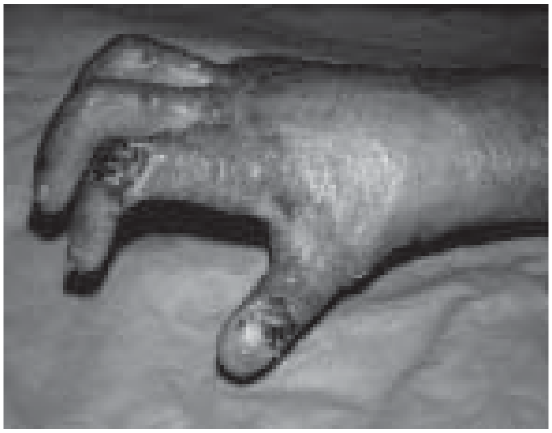


Fig. 10-44. Classic burn claw deformity.

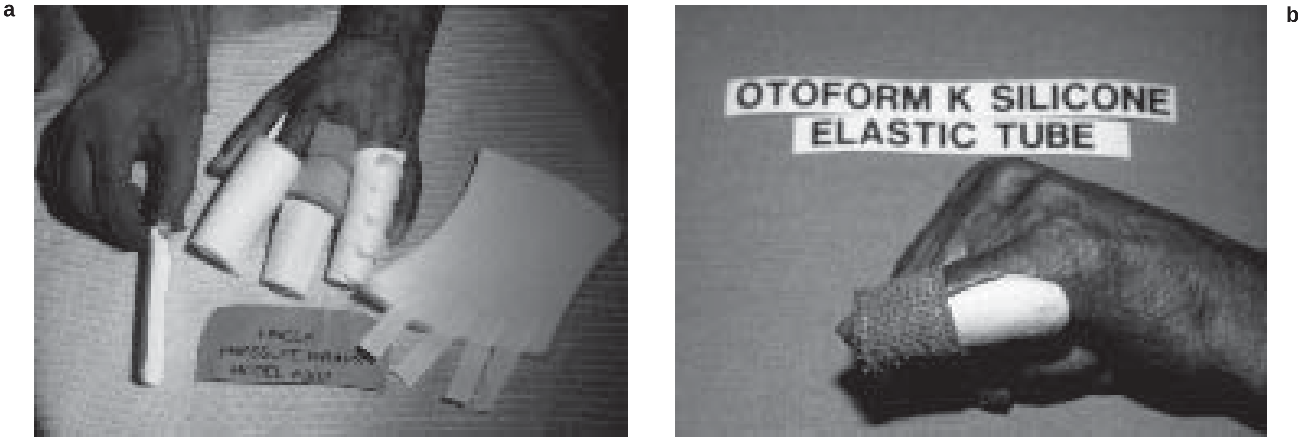


Fig. 10-45. Finger splint secured with external vascular support. (**a**) Finger pressure wraps. Note finger trough being held in patient's right hand. (**b**) Silicone finger trough secured with elastic tube.

ture band in any specific direction, the splint is changed to stretch and put direct total contact pressure on that specific collagen band. Serial casting becomes the static modality of choice.[71-73] Quick, easy off-the-shelf burn splints are advertised commercially but are rarely indicated. These rarely fit. They often block MCP flexion; the thumb web is not adequately radially abducted so the thumb web space is not maintained; the plastic is not adequately rolled away at the wrist, potentially impinging on the excised radial thumb area; and the plastic is often not rolled away from the hypothenar area to allow proper relief.

Early burn splints are secured with a bias gauze wrap such as Kerlix (Kendall Healthcare Products Co.) (see Figure 10-41) wrapped from the most distal end to proximal in a gradient manner. Securing an early burn splint with straps is contraindicated since straps do not allow for changing edema, often compromise circulation, and do not evenly distribute pressure over the edematous extremity. Elastic wraps are rarely used in the early phase to secure an orthosis or to distribute pressure because the fragile epithelium can be damaged by nongradient wrapping. When a casualty says his elastic wrap, an orthosis, or Unna (ER Squibb & Sons) dressing is too tight, or if there is any pain or numbness in the wound or distal anatomy, he must be seen and the device removed, revised if necessary, and replaced. Ignoring a casualty's complaint that leads to a pressure sore or tissue loss of any kind is unacceptable. When redness or sores are observed by the physician, nurse, or patient, the therapist should be notified and a heat gun used to flare out an edge that is rubbing or bubble out an area that is creating too much pressure. Pressure areas can cause nerve damage or convert partial thickness skin injury to full thickness damage. Padding a pressure area only increases the pressure. When adjustments increase comfort, the casualty's concurrence for splint wear increases. Tape is rarely used on splints or to secure them because it becomes sticky when in contact with drainage or topical medications, traps bacteria, may dislodge healing epithelium, and is difficult to clean.

Digits begin to contract as hand wounds are healing. Thermoplastic or silicone troughs may be applied to fingers to decrease edema and straighten the joints (Figure 10-45). These may be worn in conjunction with the resting hand splint. LMB (North Coast Medical) foam finger pressure wraps are another extension alternative. Interdigital web spacers of soft material such as Webril (Kendall Healthcare Products, Inc.) is often begun at this early stage if the webs are healed (Figure 10-46). Frequent splint readjustments are needed in this phase as the edema usually decreases rapidly with proper elevation.

The Iowa City, Buckner Microstomia Prevention Appliance (MPA) can be used to provide prolonged horizontal stretch to the mouth overnight, for several 15-minute periods, or for hourly 5-minute periods during the day (Figure 10-47).

A custom formed foam sandal or the Parkland shoe[74] may also be needed for comfortable early ambulation. If foot drop is noted, an ankle foot orthosis (AFO) is often used as an adjunct to positioning and ambulation to preserve ankle dorsiflexion. The foam off-the-shelf AFO is often adequate for ankle management if the calf is not unusually edematous or large.

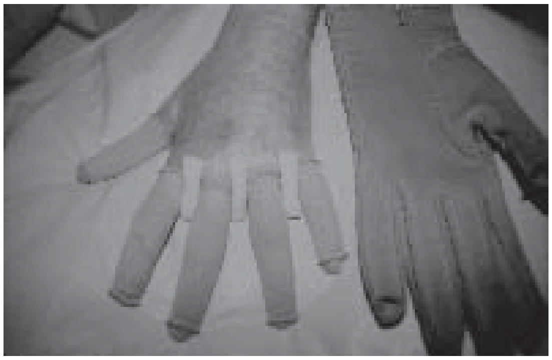


Fig. 10-46. Isotoner gloves. Felt interdigital web spacers and individual Tubiton finger sleeves are worn under the Isotoner.

Splint wearing schedules vary depending on the type of splint and the wound condition. An antideformity positioning splint is usually worn 24 hours a day if edema is present, if tendons are exposed, when the casualty is comatose, or is uncooperative with exercise. As wounds heal, splint wear is decreased to nighttime and rest periods so that the casualty may use his extremities during the day.[68] When needed, functional splints are worn during the day to facilitate independent daily living skills. These orthoses are discontinued as quickly as possible to prevent dependence and quickly increase independence. The wearing schedules should be updated daily by the therapists and nursing staff and made available for the patient, family, and physicians. Commercially available and commonly used orthotic materials are found in Table 10-4.

Precautions to observe during orthotic use include monitoring the fit to prevent unnecessary pressure sores or discomfort to the casualty and observing protruding digits for circulation, motion, sensation, and temperature (CMST). If the digits are blue or purple and cold to the touch, or blanch and refill is impaired, the device is too tight and should be removed immediately. An additional precaution is observing the skin for any areas of breakdown caused by pressure from the device or friction from casualty's movement. When the orthosis is first used it should be checked within 2 hours. If the device is not causing any problems, the time can be increased gradually, but at a minimum, the skin needs to be inspected daily. If open areas are observed, a nonadherent contact layer such as Adaptic (Johnson & Johnson) or Xeroform (Chesebrough Ponds, Inc.) is used under the orthosis. Open areas often heal more rapidly under a cast, where the casualty and staff cannot disturb the fragile epithelium. However, ointments under a cast may slow reepithelialization, and, therefore, the use of antimicrobials must be discussed with the physician.

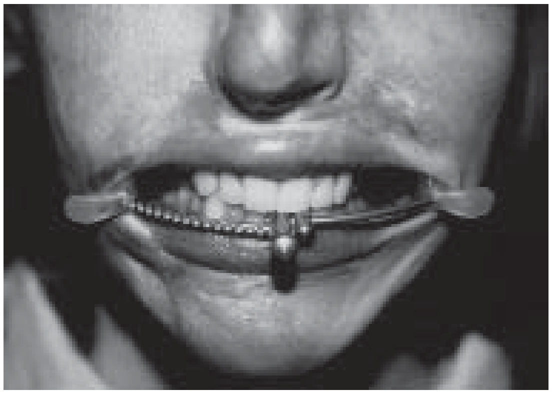


Fig. 10-47. Iowa City Buckner Microstomia Prevention Appliance.

The deleterious effects of prolonged immobilization of the synovial joints demonstrated by numerous orthopedists,[75] was the basis for the development of CPM machines. In 1988, over half the burn therapists participating in the OT/PT special interest group used CPM devices with their burn population[62] (Figure 10-48). There are two basic CPM designs: One is the anatomical design that moves the joint in an arc of motion as similar to actual anatomical movement as is possible with a machine. The other design is free linkage that provides motion to adjacent body parts, such as the forearm, and allows the joint such as the elbow or shoulder to move as it is able.

Anatomical motion is probably more comfortable for the patient. Most of the machines can be set to pause at the end range and resemble slow, prolonged stretch. Machines may be portable, freestanding, or attached to a bed or chair. Those who are most likely to benefit from use of CPM in addition to customary physical and occupational therapy include individuals who have burns involving multiple joints; comatose patients; and patients who refuse active motion because of pain, swelling, or anxiety. There are many brands of CPM machines and one is usually rented from a medical supply provider for use a month at a time.

The hand CPM device is effective in restoring hand range-of-motion when supervised by an experienced therapist. It does not damage skin grafts or newly healed tissue and the pain experienced is

TABLE 10-4
COMMONLY USED ORTHOTIC MATERIALS

Commercial Name	Usage
Polyform	Conforms perfectly to all indentations and projections in "hard to mold" areas such as the thumb. Works best for gravity assisted positions because of its draping ability. Splints must be placed in exactly the same position each application because of comformability. Conformability keeps splint from applying pressure over a scar. Cannot be remodeled.
Ezeform	Does not conform to body parts as exactly as polyform. Drapes well for both gravity and antigravity positions, but is more stretchy. Can be heated and remolded easily. Good for hand or wrist based splints that may require frequent readjustments. Because it is a rigid material, it is stable during a prolonged stretch.
Orthoplast	Best for large splints as it doesn't drape or overstretch. Can be applied to the extremity with an elastic wrap. Good for axillary conformers, ankle foot orthoses, or elbow splints.
Polyflex	Good for circumferential splints such as the elbow hinge splint because it is flexible and has some memory, making it easier to remove from the extremity.
Aquaplast	Good for small finger splints because it is light, durable, and thin.
Fiberglass	Light, strong, good for walking casts or casts which will need strength. Does not conform well to small digits or scars. Makes very little mess, sets up fast, and is durable. Rolls come in various widths so can be used for small and large casts.
Plaster strips or rolls	Drips when mixed with water. Allows more time to mold the cast properly because it sets up slower than fiberglass. Plaster casts are heavy.

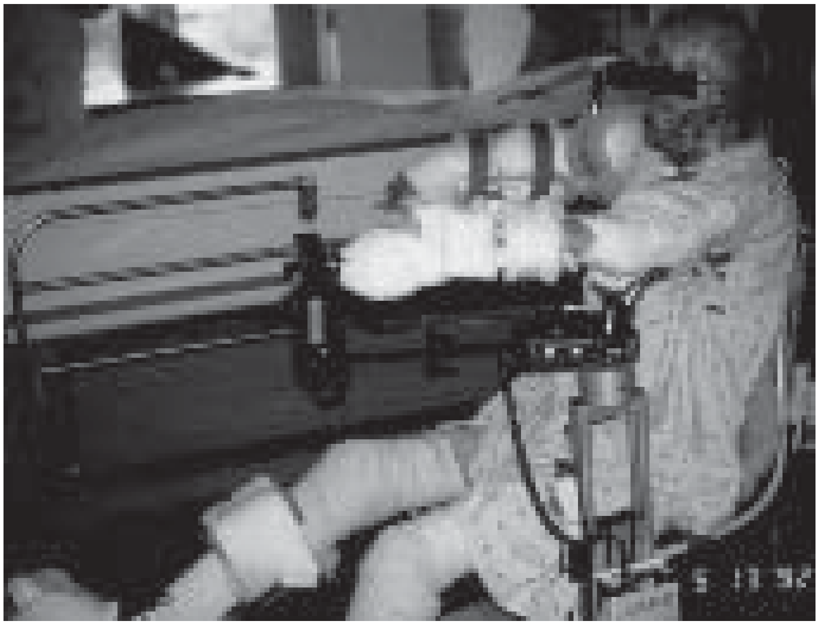


Fig. 10-48. Continuous passive motion machine during acute phase. Patient simultaneously using right shoulder, left elbow, and right knee devices.

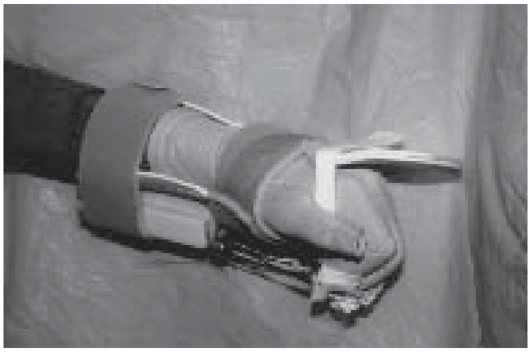


Fig. 10-49. Hand CPM device during acute phase. An Isotoner glove with Velcro strips is used to secure fingers.

the same as that with conventional hand stretching and exercise therapy[76] (Figure 10-49). Present models that include the thumb are difficult to position in a way that the thumb does not rub against the index finger. Finger motion alone is adequate to decrease hand edema. Adaptations can be made to block MCP or PIP motion and some splints can be used over the dorsum of the hand to achieve improved composite MCP and IP flexion.

One model of the shoulder CPM that attaches to the bed and moves the shoulder through 180° of flexion is helpful in preventing axillary scar bands, as well as in nourishing the shoulder joint cartilage. Another model moves the shoulder in a figure-8 pattern when the patient sits in a special chair but does not move the shoulder joint beyond 100° of flexion (see Figure 10-48). Other brands move the wrist up a bar to increase shoulder range and allow ambulation while wearing the device.

The elbow CPM can be set for low load stretching and auto reverse, so there is little risk of trauma. The mobility of elbow flexion and extension (see Figure 10-48) as well as pronation and supination can be addressed. When set for gentle motion, this device should not cause heterotopic ossification.

Knee CPM machines used during rest periods also provide improved motion at the ankle and hip (see Figure 10-48). These CPMs are useful until ambulation is possible, or longer, if joints are resistant to contracture reduction.

Functional Activities and Adaptive Equipment During Rehabilitation

General considerations. Participation in the functional activities of daily living (ADL) (daily living skills) is a very important aspect in the rehabilitation of the burn casualty.[77] Daily living skills include mobility, self-care management of environmental hardware and devices, communication, and home management activities. These major classifications are further defined as follows: (*a*) mobility includes movement in bed, wheelchair mobility and transfers, indoor ambulation with special equipment, outdoor ambulation with special equipment, and management of public or private transportation; (*b*) self-care includes feeding, bathing, toileting, grooming, and dressing activities; (*c*) management of environmental hardware and devices includes the ability to use telephones, doors, faucets, light switches, scissors, keys, windows, and street control signals; (*d*) communication skills include the ability to write; operate a personal computer; read; type; or use the telephone, a tape recorder, or a special communications device; and (*e*) home management activities include marketing; meal planning and preparation; cleaning; laundry; child care; and operating household appliances, such as vacuum cleaners, can openers, ranges, refrigerators, electric mixers, and hand operated utensils.[78]

There are numerous benefits gained from the independent performance of functional tasks and activities. Physical benefits include improving range-of-motion, fine motor dexterity, and overall endurance. The psychologic benefits include feelings of self-reliance, improved self-esteem, and more positive feelings regarding the future. Early involvement of the individual in the planning and implementation of functional activities allows the casualty to more easily resume life roles and decreases posttraumatic disability later. Independent performance of ADL hastens the discharge from the hospital to home or a less supervised setting.

Several factors can influence the overall outcome of a functional activity program. These factors include medical status (percent of TBSA, degree and location of burns); age; degree of cooperation and motivation; premorbid physical, psychological, educational, intellectual, economic, and functional status; and social resources. Additional factors that impact independent living include memory loss from medication, pain or severity of illness, cognitive changes due to anoxia or accompanying head injury, preburn or medication-induced impaired judgment, depression, and pain. Successful programs involve the casualty and family with prioritization, goal setting, and problem solving for accomplishing functional activities.

Functional activity performance can be analyzed in terms of independence, speed of performance, and safety factors when considering how a casualty accomplishes tasks. Independence in daily living skills can be achieved in a number of ways. Burned soldiers need to perform daily living skills without adaptive equipment, using repetitions to achieve improved strength and endurance. When adaptive equipment is used in the earliest phase of rehabilitation, it should be discontinued as soon as possible. Recommendations for usage of adaptive equipment, alteration of the task, employment of adaptive techniques, or modification of the environment are provided as appropriate by OTs.

Functional Activities and Adaptive Equipment During the Acute Phase

Precautions to observe during the acute stage include maintenance of good skin integrity; preservation of exposed tendons; prevention of edema in dependent extremities; avoidance of increased shoulder, elbow, and hand contractures from using adapted equipment; and prevention of pain exacerbation.

Self-feeding is encouraged despite hand burns. Often, conventional silverware with an elastic roll to enlarge the handle, if needed because dorsal hand tendons are exposed, and an elevated table are adequate to encourage self-feeding (Figure 10-50). Adaptations for self-care are discontinued as soon as possible to avoid dependence and to increase hand and upper extremity range of motion. However, adaptive equipment facilitates self-feeding for the casualty in need of it, such as one who is burned more than 70% or an electrical burn at the acute stage of burn rehabilitation. Adaptive feeding devices include long handled silverware, utensils with built-up handles (Figure 10-51), and a universal cuff for a utensil, or a roto cuff with utensils. Universal cuffs with Velcro or D-ring attachments allow independent application. Expanded universal cuffs can be fabricated for bulky dressings, casts, or splints. For the individual with severe limitations of motion, additional devices such as bent-angled silverware are available to use if wrist radial deviation is permanently limited. Utensils with extensions can replace mobility of the trunk or neck as well as decreased shoulder or elbow flexion. Utensils with a swivel attachment (Figure 10-52) and utensils with horizontal handles replace forearm supination. Vertical handles can be used when the forearm is fixed for midposition. A rocker knife can be used for cutting when a person is permanently unable to use one hand.

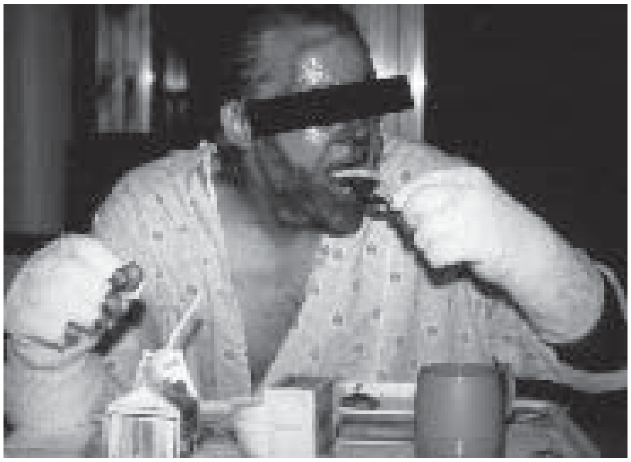


Fig. 10-50. Encourage independence in self-feeding without adaptive equipment. Here the casualty is using an elevated table.

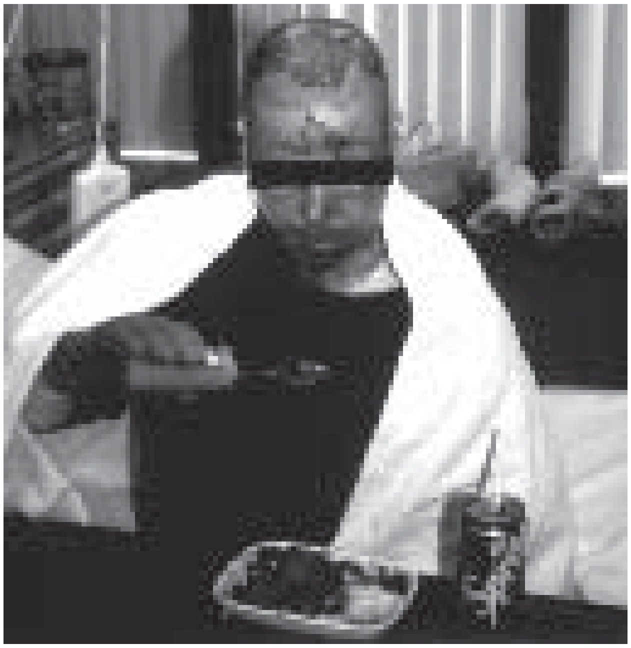


Fig. 10-51. Adaptive feeding utensil: a built up handle. Dycem is used on the table top to create a nonskid surface.

The following are a variety of aids for drinking: a long straw held in place by a straw clip (Figure 10-53), adaptive mugs and glasses, easy grip mugs with protruding handles, glasses with a cutout area by the nose, and bilateral glass holders. Sealed mugs are also helpful when a casualty lacks hand control. If a tremor is present, a weighted mug can be used. In cases when there are inhalation injuries resulting in swallowing difficulties, vacuum flow suction mugs can control the rate at which liquid is released to prevent choking.

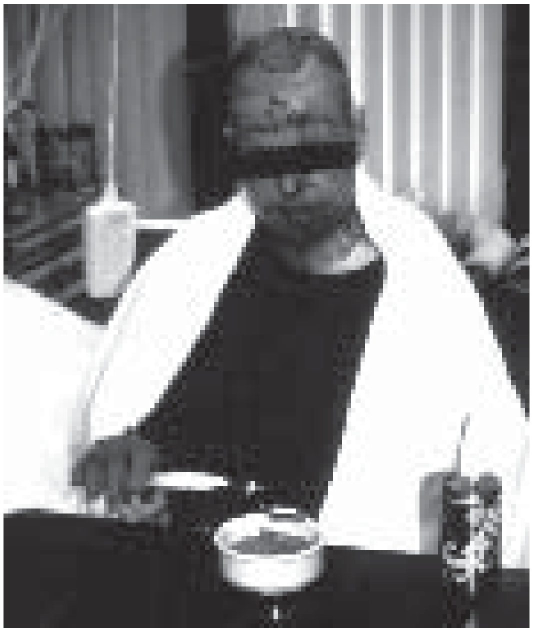


Fig. 10-52. Adaptive feeding utensil: a swivel attachment. This is the same table shown in Fig. 10-51 with a Dycem nonskid surface.

There are several types of adaptive equipment that are used for stabilization when a casualty has the use of only one hand. Dycem (Dycem Inc.) is a nonskid surface that prevents a plate or bowl from slipping when placed on it (see Figures 10-51, 10-52, and 10-53). Scoop dishes or plate guards attached to plates provide stability for utensil usage when only one hand is available for self-feeding.

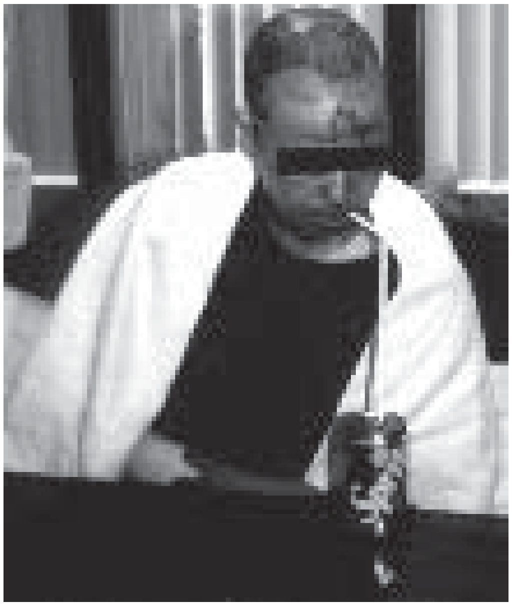


Fig. 10-53. Adaptive drinking utensil: a long straw. Again Dycem is used to create a nonskid surface

Grooming is another self-care activity that can be performed early. Oral care and hair brushing and combing are initiated when bathroom privileges begin. Although rarely needed, a built-up handle or universal cuff can compensate for the decreased ability to grasp the toothbrush. A temporary extended handle can ease teeth brushing while range-of-motion improves. Toothpaste tube squeezers are available if fine motor dexterity is limited. Denture brushes with suction cups are useful when only one hand is functional.

Hair brushes and combs can also be adapted with built-up handles and universal cuffs. Extended handles (straight or bent-angled) are useful when shoulder flexion or shoulder external rotation or both are lacking.

Communication is a functional activity that is very important in all phases of rehabilitation. This is a broad category that includes expression, reading, use of environmental controls, writing, and telephone and typewriter/computer usage. Large button, programmable phones can be adapted to have a speaker, allowing early independent telephone use. Because telephone usage keeps burn casualties in touch with their support systems, adaptations are requested early. A goose neck adjustable phone holder allows a person to speak and hear without holding the telephone. The goose neck holder affords more privacy than the speaker phone. There are special phone holders for weak grasp. A phone flipper is a lever that can be added to the base of the telephone to activate the connection button if a casualty cannot depress the button. This can be operated by hand or by mouthstick. Another type of phone adaptation is a touch-tone phone adapter. This is a device that enlarges regular push buttons. It can be operated by a very light touch.

In the acute phase of rehabilitation, expression assists the casualty to interact with others in the environment and decreases fear and frustration. In severe burn cases, a casualty may be intubated, ventilator dependent, and unable to speak. If there is prolonged exposure to smoke inhalation, vocal cord injuries can result and speech can be unintelligible or contraindicated. Communication boards composed of letters, pictures, body maps, words, or complete sentences can be used to convey thoughts if the casualty can point. There are also sophisticated communication devices such as the Voicaid or Prentke Romich Touch Talker or Light Talker (Prentke Romich Co.) that have voice outputs. These can be operated by touch or by a light-activated beam. However, casualties are often too ill or medicated to use complicated communication devices.

There are a great number of environmental controls that can be adapted for independent usage. Call lights, bed controls, lights, and other appliances can be operated by environmental control systems. Environmental controls are available in a wide variety of switching systems with specific mounting devices. Examples of switches are the basic plate, light touch plate, air cushion, rocker switch, foot switch, wobble switch, sip and puff control, and the joystick. (The same switch and input device method can also be used to operate a communication device, computer, or electric wheelchair in other phases of rehabilitation.) The switches can be specially ordered from companies such as TASH (Prentke Romich Co.) or can be purchased from Radio Shack.

Reading can be a very important functional exercise when a casualty is confined to the bed (Figure 10-54). There are several types of book and magazine stands available if needed. Bed readers, positioned slightly overhead, are useful when an alert person must stay in supine positioning. A prone person can use a floor stand or music stand with pages stabilized by clothespins. Book stands, height and angle adjustable, clamped to a bedside table or headboard provide optimal positioning. Books or magazines can be placed on a foam wedge. Book stands with automatic page turners or a mouthstick can be used if a casualty is unable to turn pages independently. Prism glasses allow a person to look down at pages to read while the head is positioned forward. In cases of preexisting visual acuity deficits, a variety of magnifiers can be used in addition to large print books.

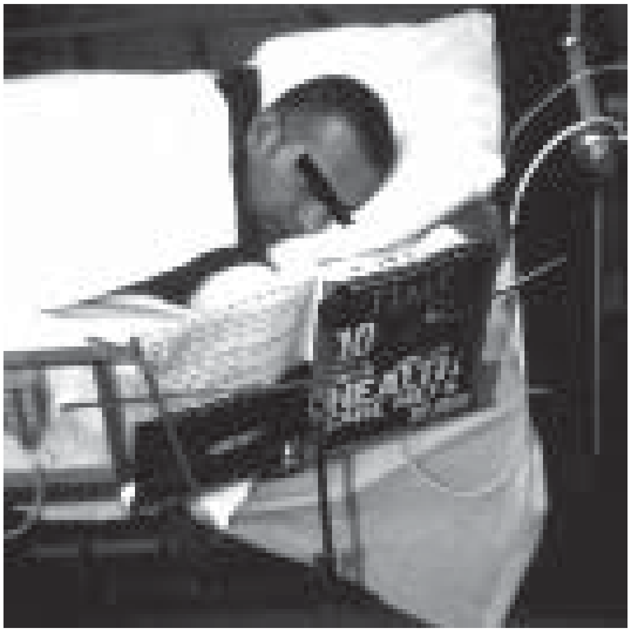


Fig. 10-54. Reading for recreation and exercise. The casualty is able to read by using a reading stand.

Writing is another important self-care activity that can be performed in early stages of burn rehabilitation. Pen and pencil holders are useful temporary adaptations when a casualty's ability to grasp or pinch is decreased. Built-up foam handles or plastic easy grip adapters facilitate holding the pen. Special splints or adaptive writing utensils can be fabricated out of thermoplastic materials, or commercially available splints can be obtained to customize writing devices. A clipboard can be used to stabilize the paper when the nondominant hand is unable to hold the paper steady for writing. A writing device attached to a mouthstick can be used if both hands are nonfunctional for writing.

Pain Management in Burn Patients

Pain is purely subjective, difficult to define, and often difficult to describe or interpret. It is defined as an unpleasant sensory and emotional response to a stimulus associated with actual or potential tissue damage.[79,80] It is a multifaceted experience extensively influenced by anxiety, depression, and expectation.

Acute pain is a biological symptom of an apparent nociceptive stimulus, such as acute tissue damage or trauma. It lasts as long as the tissue pathology lasts, is generally self-limiting, and as the nociceptive stimulus lessens, the pain decreases. Acute pain means that the patient's pain seems to match in time with what one knows about his injury. A patient with severe burns may have pain that lasts for months, and by examination of the patient's skin there is still evidence of tissue damage. Therefore, he is still having acute pain. The real differentiation between acute and chronic pain is whether the patient's complaints can be matched to the understanding of mechanisms of tissue damage, not the actual duration of the pain.[81] Everyone has experienced a burn from a hot skillet or hot water, so one can only imagine the pain caused by a major burn.[82] Burn pain has been found to correlate with depth of burn and location but not the severity or extent. Also, it is well known that burn pain severity can not be predicted on the basis of age, gender, ethnicity, education, occupation, history of drug or alcohol abuse, or psychiatric illness.[83] Nurses have been shown to administer analgesics in less than the maximum allowed dosage and yet complain that the physician is not prescribing adequate analgesia.[83] Nurses have both overestimated and underestimated the patient's pain, and have a tendency to overestimate the degree of pain relief from medications given. Emphasis has been placed on the implementation of systematic procedures to assess pain.[84]

The goal of pain treatment is to try to eliminate pain. However, to abolish pain without producing side effects is difficult if not impossible to accomplish. The successful rehabilitation of a burn casualty depends on active participation of the person in the treatment program. The casualty must be relatively comfortable, that is to say, his pain must be at a tolerable level if the needed active participation is to be achieved. Yet the most intense pain does not relate as much to the burn injury itself as it does to the "trauma" caused by the therapeutic procedures. When patients are at rest, the pain may be relatively mild on the average. In contrast, when the casualty undergoes therapeutic procedures, significantly more pain is experienced and its intensity can reach extremely high levels.[85] Also, the pain experienced does not necessarily lessen over time. Studies have shown no correlation between patients' self-reported burn pain scores and the elapsed time since injury. Although there is an interrelationship between depression, anxiety, and pain, it has been demonstrated[85] that, in burn patients, high levels of anxiety did not necessarily associate with high pain scores during therapeutic procedures.

The pain management should therefore address the background level of pain as well as the breakthrough or procedural pain. The goal should be a tolerable level of discomfort and active participation of the casualty in the rehabilitation program.

In considering the prescription of analgesics, the physician should consider the duration of action, the route of administration, and the dosing schedule. Morphine is the standard drug against which all other analgesics are measured. No other opioid has been shown to be superior to morphine in analgesic effect. All opioids can be administered parenterally, orally, or rectally. Fentanyl ([Fentaugh] Janssen Pharmaceuticals Inc.) can be administered transdermally as a 72-hour skin patch. Parenterally administered opioids have 100% bioavailability, while oral bioavailability varies between 15% to 25% for morphine and 69% for codeine, methadone, and oxycodone. Also, the liver metabolism varies considerably in the burn patient as his acute medical management proceeds. There is correlation between plasma morphine levels and pain relief and there is no known reason to not use opioids in appropriate dosage schedules in burn patients to control pain.[86] The half-life of morphine oral solution was found to be 3 hours, while the half-life of M.S. Contin (Purdue Fredrick Co.) was 14.7 hours. Time to peak levels of morphine sulfate was 30 minutes, and 1.4 hours for M.S. Contin. Therefore, breakthrough or procedural pain may be controlled with rapid release oral morphine, and sustained release morphine is a good choice in the management of the background pain in burn patients on an 8- to 12-hour dosage schedule.[86] When pain control during the acute phase is inadequate, the experience is unnecessarily traumatic, with psychological and physical difficulties later. Even after the pain is eventually controlled, the patient has been conditioned to diminish effort and movement and, therefore, exercise and repetitive movement crucial to maximum function is not achieved.[87]

Anxiolytic therapy, in addition to analgesics, may well help the burn casualty attain a satisfactory comfort level. Benzodiazepines are the drug of choice for this management. The aim of sedation is to reduce restlessness, anxiety, and fear. The use of benzodiazepines in conjunction with morphine is more effective in controlling pain and anxiety then using one agent alone. Versed ([Midazolzam] Roche Laboratories) has a rapid onset of anxiolysis and amnesia and is gaining popularity for acute burn procedures. In the elderly it should be used cautiously. Cardiovascular monitoring is suggested initially since it can cause cardiac arrhythmias (and respiratory arrest). Intranasal administration is possible because of its neutral pH and absorption across the nasal mucosa.

Inhalation analgesics have been shown to be very effective and safe in a large series of more than 800 patients during more than 1,400 treatments with less than 5% side effects in the patients and no long term side effects to patients or staff.[88] During the acute phase, if the patient is on a respirator, depression of the respiratory center is less of a concern and more analgesia can be given at times to allow tolerable levels of discomfort during rehabilitative therapeutics.

Hypnosis with burn cases has been reported as effective in some patients with reports dating to 1965.[89] The studies are difficult to control since other treatments such as analgesics are used simultaneously; however, most report hypnosis as a useful adjunctive treatment. The contraindications to hypnosis are (*a*) if the patient is not interested in it, and (*b*) if the patient has a large secondary gain component to pain.[89] The person administering the hypnosis must know the patient's allergy history to avoid stimulating allergic reactions during suggestion. Relaxation techniques and self-imagery are at times very helpful as an adjunct to rehabilitation procedures and are recommended since adverse side effects are almost nonexistent. Group and individual psychotherapy is also effective in reducing pain behavior in some patients. Individual psychotherapy is needed more commonly in patients with previous maladaptive behavior.[82]

Use of transcutaneous electric nerve stimulation (TENS) has been attempted in burn patients. There is at least one controlled study[90] of conventional TENS being as effective as morphine in the acute stage of burn care during Travase debridement. In general, the burn injury and the pain would need to be quite localized for this treatment to be effective and, therefore, it is used only occasionally. There is one report[91] of effective use of TENS as auricular acupuncture to reduce pain in burn patients with less than 35% TBSA burn. Since the side effect of TENS is limited primarily to skin rash, this treatment modality should be further investigated and utilized when the conditions so indicate.

Psychosocial Aspects of Burns During the Acute Phase

Intervention to diminish maladaptive emotional reactions of burn casualties is an important aspect of burn treatment. Adjustment to the burn injury is influenced by the severity of the burn, location of the burn, age, sex, preinjury psychosocial stability, educational status, hospital and outpatient treatment environment, and by community and family support systems.[92] The burn casualty's ability to cope with the stresses of his situation are influenced by his past experiences and by the awareness, understanding, anticipation, and response of the team managing his care.

Burns sustained during a combat situation are usually accompanied by the psychological effects of battle stress. Multiple other physical agent wounds may be experienced at the same time. The depth of the wound as well as the location will determine whether the casualty will return to duty or be evacuated. A nonbattle burn injury may be the result of accidents secondary to battle stress conditions that reduce performance, making the soldier vulnerable to injury.[93] Sleep loss is one of the most significant contributing factors to nonbattle injury. When under stress, the soldier's mental abilities are affected, including alertness, attention to details, perception, reasoning and comprehension, memory, motor responses, communication, self-control, and interpersonal relations. Soldiers with minor burn injuries, especially if separated from their units and treated in the rear, have psychological reactions that retard their recovery unless they are also treated with the same intervention provided for a battle stress casualty.[94] Shock, numbness, and detachment are commonly noted at the time of injury, which protects the person while he collects emotional resources to cope with the injury.

The principles of battle fatigue prevention are taught to prepare soldiers who will face the stress of battle and, if burned, the additional stress of injury. The routine training of soldiers in stress control principles, including rapid relaxation techniques, is valuable in the event of a burn injury. Coping with the stress of battle involves balancing internal reactions to meet and overcome external stressors. When the external stressors of sustained battlefield conditions cannot be removed, the soldier must learn to use stress reducing strategies to cope. These techniques are taught by leaders and practiced with the use of the buddy system to reinforce learning. In the event of injury, the soldier's mind-set for recovery may be positively affected by what leaders and buddies say and do.[95]

The mental health section at the unit level can provide support that focuses on mild and moderate battle fatigue and burns such that the soldier can return to duty. For the minor burn injury of less than 1% TBSA, in which return to duty will occur as soon as the wound heals, the soldier needs to be treated using the principles of proximity, immediacy, and expectancy. Keeping the soldier as close to the action as possible reinforces his identity as a soldier, rather than a casualty. The farther away the soldier is from his primary group, the more difficult it is to have him return to normal duty. Immediacy is important in helping the soldier to cope with the situation as early as possible and to identify battle fatigue symptoms as they appear. Expectancy means to expect the soldier to return to normal duty where his comrades need him. Treatment using the principle of expectancy is appropriate not only for the soldier returning to duty within a short recuperation time, but also for the soldier who will require extensive burn rehabilitation.

For the soldier with severe enough burns to be evacuated, early psychological intervention and support is a key factor in obtaining motivation and cooperation with treatment. The burn casualty will be separated from his unit and does not have ready access to family or friends for psychological support. An important role of the treatment team at all echelons includes providing emotional support and reassurance to reduce the anxiety and fear experienced by the soldier. The evacuation team provides ongoing orientation information, including calling the soldier by name. Part of the chaplain's mission is to provide comfort, assurance, and encouragement to the soldier. The provision of positive affirmations and encouragement even during initial delirium reinforces his internal will to live.[96] The speed of physical recovery may be affected by the early emotional adjustment to an acute burn.[97] This adjustment is facilitated by keeping the soldier informed about what is happening to him. Providing preparatory information prior to intervention procedures and encouraging the use of simple relaxation techniques during treatments contribute to emotional well-being and ability to tolerate pain during this acute stage. It is important to repeat instructions, provide frequent orientation, and if possible, allow verbalization of fears.

The immediate reaction of a massive burn casualty includes psychological shock. This reaction usually lasts a short time and may involve disorientation delirium, emotional instability and lability, and sleeping problems with nightmares of being burned.[92] Fear and anxiety also accompany the initial part of the acute phase of burn recovery. The soldier should be allowed to express his fears regarding death. These fears should be met with a reality oriented response regarding his chance of survival.[98] Behavioral manifestations, such as increased startle response, difficulty in concentrating or following instructions, withdrawal, resistance to treatment, overt hostility, or other inappropriate behavior, are often noted immediately after the burn.[99] The burned soldier may or may not realistically perceive a major threat to his survival. Information regarding the seriousness of the burn is given, if possible, before pain medications cloud awareness.

Treatment team members need to obtain information regarding the soldier's background, interests, personality, and family and unit relationships. This information is useful in developing a plan to deal with the immediate psychological responses as well as approaches that will be most effective in helping him cope with a prolonged, rigorous recovery. The early inclusion of the family in the rehabilitation process is very important to the adjustment of the soldier and family. The Red Cross may be helpful in relaying information between the soldier and his family while they are separated as well as assisting the family in securing quarters near the medical treatment facility that the soldier returns to for treatment. Intact and supportive families have been influential in successful long-term psychosocial adjustment of survivors of severe burn injury.[100] Family members may be interviewed by the social worker, nurse specialist, psychologist, psychiatrist, or chaplain to get an accurate history of the casualty's preburn personality, coping styles, and reactions. This will add to the information from the soldier to facilitate development of strategies in managing the rehabilitative care of the casualty.

It is important to involve the family in a support group and provide educational classes to inform them about burn injuries and treatment. Through interactions in the group, family members have the opportunity to learn from one another and support each other. Family members are often responsible for immediate communication with girlfriends, boyfriends, and community groups or churches. Family and friends at home can provide significant support and focus toward the future by letter writing and maintaining positive contacts with the burned soldier.

Following the resolution of the initial shock of the burn incident, the casualty becomes more aware of the impact of his burn. Orientation improves and survival anxiety diminishes. Thoughts are more focused on concerns about oneself, including the effects of changed appearance and altered function or life-style. The soldier's preburn physical, emotional, intellectual, social, and spiritual nature provide his initial coping skills. Additional skills are usually needed. Psychologists, social workers, or chaplains help the casualty to focus on regaining as much control as possible and in redefining the meaning of the accident, desensitization from the reminders of the injury, dealing with stress in a positive way,[101] and in gradually accepting loss and trauma as a matter-of-fact part of the past. "Getting their feelings out" is discouraged[102] to preserve fragile but useful coping mechanisms, including denial.[103] Adequate pain control, correction of sleep disturbances, decreasing the fear of long-term consequences, and cooperation with the burn team become crucial for optimal outcome.[92] Numerous interventions are appropriate and may include (*a*) providing as much physical comfort for the casualty as possible; (*b*) providing ongoing orientation information including calling the soldier by name; (*c*) mentioning the date and time of day; (*d*) providing explanations about the procedures that are being used, even if the casualty is comatose; (*e*) providing relaxation training after orientation is established; (*f*) providing routine for bathing time, exercise, and meal times to decrease unexpected procedures; (*g*) encouraging family involvement as soon as possible; and (*h*) emphasizing individual control over as many situations as possible.

Losses and changes related to the burn injury affect each soldier differently. Growth, finding new strengths and coping mechanisms, recovery from grief, and developing renewed goals or emotional growth occur sometime after the soldier realizes the impact and disruption he will probably experience from the injury.

When the soldier determines that there is potential for survival, his focus may turn to the perceived pain and its alleviation. This focus frequently results in increased reports of pain as well as requests for analgesia. When adequate pain control is not provided, the casualty begins to believe that pain will be associated with each treatment and may result in poor compliance with treatments. The anticipation of pain may be complicated by having painful procedures done at different times, thus increasing the anxiety level. Adequate medication dosage for pain, anxiety, and sleep is increased because of the hypermetabolic state of the casualty. When IV lines are in place, adequate PCA to allow activity during the day, interspersed with comfortable rest periods, improves cooperation with antigravity positioning and elevated exercise that ultimately also result in diminished pain. Relaxation training, counseling, and behavioral management are effective nonpharmacological ways to deal with pain. However, they never replace analgesic, hypnotic, or tranquilizing medications. The goals of pain management are to maximize comfort, minimize disruptive behavior, and increase cooperation and productivity.[92] When complaints of dysesthesias and pain are misinterpreted by the staff as manipulative behavior at about the same time pain medications are being tapered, cooperation with therapeutic modalities is undermined. Short continuance of narcotics combined with desensitization techniques will undoubtedly reduce the problem. Most neuropathies resolve slowly. However, in addition to being taught desensitization and compensation techniques, the patient will appreciate assistance with reintegration of sensory information. Benson's relaxation[104] (Exhibit 10-3), practiced daily, will help the person tune out sensations he would not have been aware of before the accident. For some casualties, dysesthesias from the healed tissue become a central, compelling part of their awareness. Individual counseling as well as reassurance by the physician and therapist help the soldier accept the return of sensation as a positive sign, even if it is temporarily distorted and therefore painful. Patients slowly come to realize that the skin emergency has passed and by using vision as well as tactile sensation from the burned and unburned parts, the sensory information will be more quickly reintegrated.

As orientation improves, additional behavioral methods of relaxation may by implemented. These include Benson's relaxation response, autogenic training, biofeedback, imagery, and distraction. Other behavioral interventions include deep breathing exercises, which may alter sensation in a negative way, especially if the casualty hyperventilates when attempting deep breathing; and progressive muscle relaxation (although this is painful if the overlying skin is burned and therefore can cause pain instead of relaxation). Soldiers are taught rapid relaxation techniques as a part of their training for management of stress in army operations. A combination of what the soldier is familiar with and these nonpharmacologic techniques assist the individual to be in greater control of his situation.

Immobilization Phase of Wound Care

The goals in the immobilization phase of wound healing include continuing analgesia; permanent wound closure with graft adherence; modifying edema; preventing complications; maintaining joint and skin mobility as possible; educating the casualty and family to the expected results and appearance of early skin grafts; behavioral interventions to assist prolonged bedrest and to redefine skin graft operations as positive, rather than negative, in the course of wound healing; and counseling to assist the soldier to focus on the positive final outcome, rather than on loss of independence, pain, or inactivity.

Wound and Skin Care and Nursing During the Immobilization Phase

The immobilization phase of healing after grafting can be a trying time for casualty, family, and staff alike. Casualties may see it as a setback if they are unable to exercise areas they had diligently been exercising. Thorough explanations to casualty and family about grafting and the period of immobilization will help to prepare them emotionally for the postoperative period. Postoperative positioning and splinting should be discussed with the physician, therapist, and nursing staff preoperatively. Whenever possible during preoperative teaching, the nurse should assist the casualty into the position that will be assumed postoperatively. This will

EXHIBIT 10-3

OUTPATIENT HOME CARE PROGRAM: THE RELAXATION RESPONSE

1. Sit quietly in a comfortable position. Choose a place where you will not be disturbed.
2. Practicing is best done sitting on a comfortable chair with the feet flat on the floor, the hands on the legs, and the head unsupported.
3. Close your eyes.
4. Breathe through your nose. As you breathe out, say the word, “one,” silently to yourself. Breathe easily and naturally.
5. Continue for approximately twenty minutes. You may open your eyes to check the time, but do not use an alarm. When you finish, sit quietly for several minutes, at first with your eyes closed and later with your eyes opened. Do not stand up for a few minutes.
6. Do not worry about whether you are successful in achieving a deep level of relaxation. Maintain a passive attitude and permit relaxation to occur at its own pace. Distracting thoughts are normal. When these thoughts occur, return to repeating “one”.
7. Some patients report that it is difficult to judge for themselves whether this technique is working. Often, after a week of consistent practice, they report feeling more alert and either losing their discomfort or becoming less aware of discomfort.
8. Patients also report that if they practice this procedure within two hours after a meal, the relaxation does not seem as satisfactory. Deep relaxation can often occur best just after awakening when the body is well rested and before any food is eaten. You may be familiar with increased dreaming when you have eaten a large meal just before going to sleep. That is one example of increased activity of the mind in response to digestion.
9. Also, this procedure may interfere with your sleep if done within 3-4 hours of bedtime.

Modeled on the work of H. Benson, MD, Thorndike Memorial Laboratory, Harvard University.

assist in solving discomfort issues in advance. Preoperatively, a low air loss bed or air fluidized bed should be considered, not only for comfort reasons, but also for pressure reduction on healed, unhealed, old or new graft sites, and donor sites.

Postoperatively, the nursing staff must know exactly what operative procedure was performed to maximize positioning and to avoid any potential complications. The activity level must be known and also what nonsurgical sites can continue to be exercised. Continuous elevation and checking CMST of grafted extremities is performed at least every 2 hours to prevent complications, which may include increased edema; poor circulation; or pressure caused by dressings, splints, or casts that may be too tight.

Appropriate analgesia and sedation should be administered to keep the casualty comfortable yet not oversedated during the immobilization phase. Other techniques such as relaxation, imagery, or music that the casualty found helpful in the past should again be used. Casualties may require more emotional support during this phase; the nurse can provide this support by effective listening, giving reassurance, explaining all procedures that are done, providing pain relief, and enlisting the assistance of the unit chaplain or psychologist.

Meticulous wound care in nonsurgical areas needs to be performed at least daily. Observation of the surgical areas for signs of increased bleeding, infection, or because of complaints of pain from splints or dressings should continue throughout the period of immobilization.

The first postgraft dressing change, at the direction of the physician, occurs between 1 and 5 days postoperatively. This dressing change can be done at the bedside or at the hydrotherapy area. Thorough soaking of the gauze outer dressings with water or saline will facilitate their removal without disruption of the underlying grafts and any overlay dressings that may be used to secure and protect grafts. Graft disruption is less if the dressing is bent back 180° as it is pulled away from the wound, rather than lifting it at 90°. Daily dressing changes of the graft sites are usually resumed after this time (Figure 10-55). If the contact layer is adherent over the graft, application of a fine mesh gauze impregnated with an antibiotic ointment for 1 hour prior to removal will decrease sticking and bleeding with removal. This layer is not removed until the interstices are closed, unless infection is noted under it. Evaluation to determine time for resumption of exercises is completed. Safe exercise depends on graft take, durability, and location. When it has been determined that the casualty can resume range-of-motion exercises, daily living skills should also gradually be resumed. Activity levels should be increased daily. The casualty should assist in goal setting and scheduling of activities with the assistance of the primary nurse.

Exercise During the Immobilization Phase of Wound Healing

Many contractures originate during the postgraft period when any joint underlying a skin graft must be immobilized for optimal autograft adherence. It is impossible to maintain ideal positioning at all joints. For the severely burned casualty, 3 to 7 days of immobility during skin graft vascularization severely limits active motion. When a severe inflammatory process surrounding a joint is noted preoperatively, the results of immobilization can be disastrous. However, with early grafting procedures, early resumption of supervised gentle-active motion, and carefully designed immobilizing orthoses, satisfactory outcomes can be anticipated. While the casualty waits for skin grafts to adhere, the therapist recommends an exercise program to prevent phlebitis, pneumonia, and contractures during the bedrest period. Exercise is excluded from any joints underlying the graft and one joint proximal and one joint distal to the graft. Quadriceps isometric exercise and ankle pumps are easy to teach the casualty whose leg is not grafted. However, casualties often need cueing to initiate exercise and to avoid moving grafted areas. When only the upper extremity, head, or neck is grafted and can be safely immobilized, the casualty may be up in a chair and ambulate (Figure 10-56).

It may be possible to provide positioning for the anesthetized casualty who is undergoing grafting,

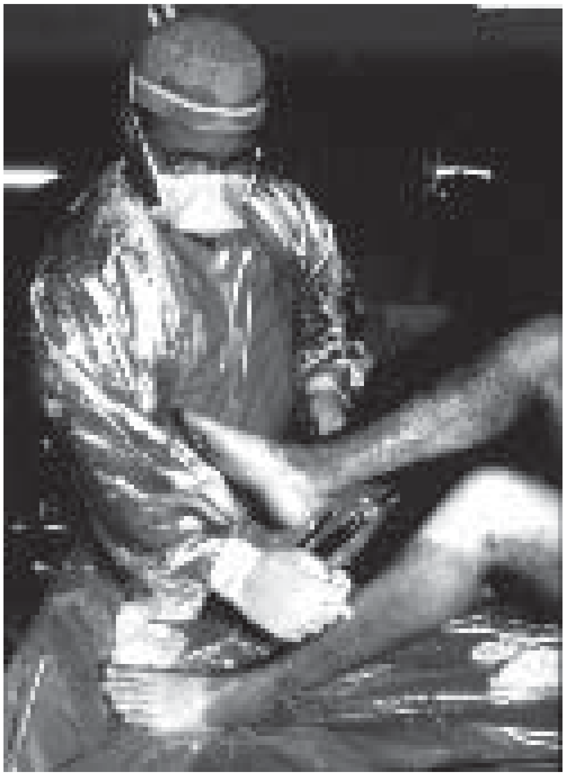


Fig. 10-55. Wound cleansing and assessment postgrafting.

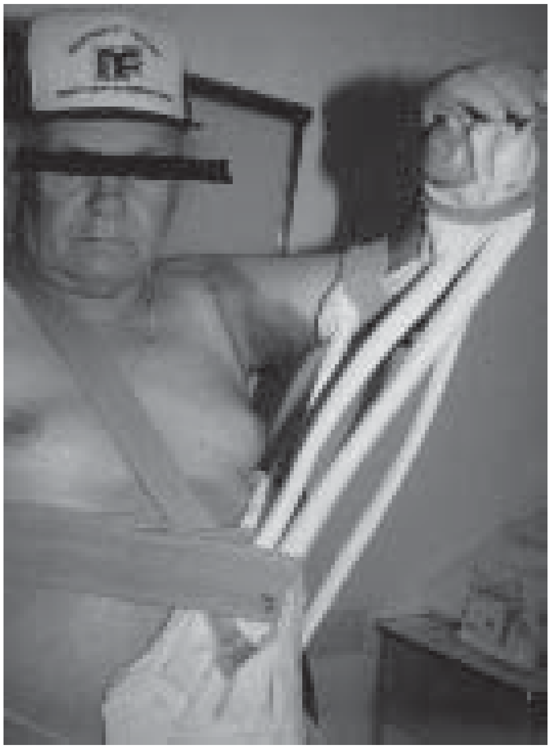


Fig. 10-56. The patient is ambulating on the first postoperative day with immobilization of upper extremity graft.

which will allow the contracted areas of skin and scar to elongate because of the plastic property of connective tissue. Excessive stretching or forcefulness can result in bleeding, swelling, and increased tenderness, so great care must be used. To stretch an ankle, for instance, it may be acceptable to place the casualty's foot on a foam block, with the hip and knee bent, and allow the heel cord to elongate slowly as the casualty lies relaxed in the supine position. The "creeping" elongation of tissue in this way results in change that may be maintained by active exercise when the casualty is awake; the slow elongation will be painless because of the anesthesia for grafting.

Therapists must adhere to the medical center's policies and procedures governing who may attend casualties in the operating room; there must be documentation of each person who enters the operating room and techniques followed during surgical procedures. The therapist who wishes to evaluate range-of-motion or provide this type of stretching when the casualty is under anesthesia must first discuss this with the surgeon. Then a protocol should be written covering the roles and responsibilities of the therapist. The operating room nurses and the person administering anesthesia will appreciate being consulted prior to the therapist participating in casualty care in the operating room.

Safety factors are stressed for everyone involved in surgical procedures. The anesthetized casualty is unable to produce a normal response to painful or injurious stimuli. Therefore, proper positioning and padding are important to avoid pressure points, stretching of nerves, or interfering with circulation to an extremity. Whenever the casualty's position is changed, it must be done slowly and gently to allow circulation to readjust. The casualty has a diminished ability to compensate for physiologic changes caused by motion or stress. The anesthetic agents are depressants and predispose the casualty to respiratory complications. The casualty's chest must be free for adequate respiratory excursion at all times and pressure must never be exerted on the chest while the therapist is holding an arm or hand. The tidal volume is decreased as much as one third when a casualty lies horizontally so constriction of the neck must also be avoided. The casualty benefits from unhindered diaphragmatic movement and a patent airway. The anesthesiologist guards the head and supports it during movement. Arms must never be hyperextended, and shoulders may not be moved above 90° to 100° of flexion or abduction.

The therapist checks with the anesthesiologist and the circulating nurse before examining range-of-motion. Motion must be checked quickly if the area is being prepared for grafting or as a donor. The therapist may "scrub in" if this has been arranged in advance. IV lines, monitoring electrodes and connectors, airway connections, and electrical equipment are always preserved. Range-of-motion evaluation cannot cause inaccessibility of the operative site. Sterile technique is not broken or the sterile field contaminated. Avoid occlusion or pressure on peripheral blood vessels, and ensure that restraining straps, if removed, are properly replaced. Bony prominences should be protected from constant pressure against hard surfaces, and prolonged compression to or stretching of peripheral nerves should be avoided. The extremities must be well supported whenever the therapist is not working with them. If injuries occur from malpositioning during anesthesia, the brachial plexus and ulnar, radial, and peroneal nerves are most frequently injured. Finally, strain on the casualty's muscles can result in needless postoperative discomfort from lack of protective muscle tone during anesthesia.

Positioning During the Immobilization Phase

The casualty must continue antigravity positioning and prevent disruption of the new graft. The team consults with the surgeon to determine positioning well before the scheduled grafting procedure. Clinitron (Hill-Rom) or Kinaire (Kinetic Concepts) beds distribute pressure; however, they promote protraction of the shoulders and thoracic kyphosis with a subsequent reduction in vital capacity. When postoperative use of the air cushion bed is recommended, high air flow cushions are placed under donor areas to facilitate drying, and low flow cushions are placed under the grafted area to prevent desiccation. The bed cushions must be changed before the person is placed in the bed postoperatively. All positioning should be tried for a night by the casualty, to solve any discomfort or daily living skill problems such as urinal use. The graft and one joint proximal and distal are immobilized. All of the positioning devices available during the early phase may be adapted for this phase. Wedge pillows or over-the-bed suspension with slings are useful for upper extremities. Deltoid aids and elevating tables also assist in alternating positions during this time. Properly positioning the body part so that the new graft is at optimal length will minimize contractures. The casualty is kept on bedrest unless immobility of the graft can be maintained

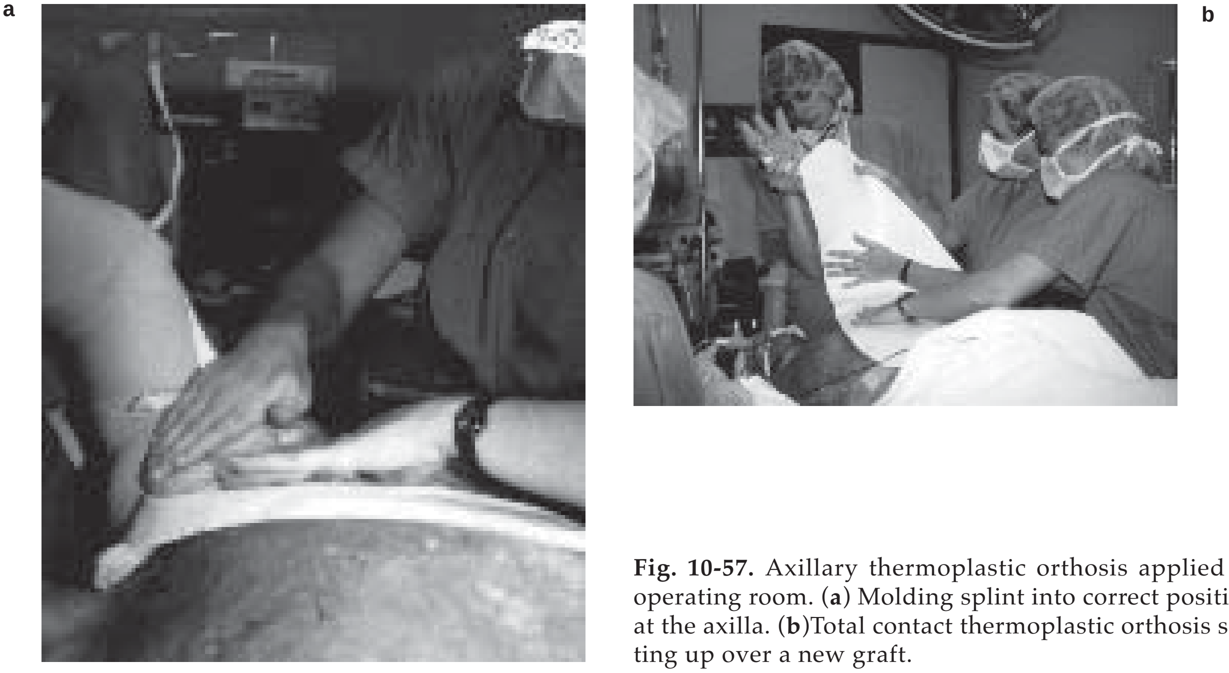


Fig. 10-57. Axillary thermoplastic orthosis applied in operating room. (**a**) Molding splint into correct position at the axilla. (**b**)Total contact thermoplastic orthosis setting up over a new graft.

while sitting in a chair or walking for short distances during bathroom trips. The body part grafted is elevated to prevent edema. If the posterior side of the body or head is grafted, the casualty is positioned prone or on an air-fluidized bed or air-flow cushion bed. In these cases, the graft must be protected with a plastic barrier, if needed, because thin dressings do not prevent graft desiccation from high air flow.

Orthosis During the Immobilization Phase

Widely varying materials and protocols have been suggested for postoperative skin graft dressings and orthoses. All have the common purposes of immobilizing and protecting the grafted area, preventing edema, and speeding wound healing. Thermoplastic splints applied in the operating room conform precisely to the graft and immobilize the extremity. (Figure 10-57) Additional considerations for orthoses are convenience, comfort, and cost.

In centers using the exposure method of graft healing, elevation, support, and protection of the grafted areas are accomplished using pillows, slings, metal and net "hammock" positioning, open thermoplastic orthoses, or skeletal traction. Safely securing a thermoplastic orthosis without impairing circulation is difficult when the burn is circumferential and extends beyond the borders of the graft. Restless casualties dislodge postoperative orthoses. If the orthosis rests on the graft it will inhibit tissue survival.

When the healed wound is expected to be especially fragile, as it is after cultured epithelial or artificial skin grafts, overhead suspension of the body part is indicated. The proper use of skeletal immobilization prevents nerve damage and joint or soft tissue strain.[105] Precautions to be addressed include use of proper counterbalance weight for each individual limb, thorough investigation of the casualty's complaints, frequent observation of the entire rope and pulley system to avoid weights resting on the floor or bed parts, lubrication of pulleys with silicone spray before attaching traction, and shielding of the weights to keep them from being bumped by passing visitors, children, and equipment.

After tangential excision using a tourniquet, a bulky pressure wrap is often used to prevent bleeding and protect the grafts. In this case, the whole extremity is wrapped in cotton batting and elastic bandages and suspended from overhead traction for 24 hours. After 24 hours, the bandaged extremity is elevated on an arm wedge or the foot of the bed is elevated.

To make a bulky wrap, a single layer of nonadherent dressing is used in contact with the graft. An example of this use is in a hand burn. A contact layer such as Biobrane (Winthrop Consumer Products) is stapled in place over the grafts. Wet fine mesh gauze is wrapped around the contact layer. An adequate number of completely opened gauze 4 x 4 in. sponges are placed into the palm, between the interdigital webs and into the thumb web space.

A Kerlix (Kendall Healthcare Products Co.) roll is placed into the palm to increase the thumb web space. Fluffed gauze pieces are placed over the dorsum of the hand and thumb with a snug Kling (Johnson & Johnson) wrap. The entire wrap is covered with cotton batting and secured with a gradient elastic bandage wrap. Gradient means progressively decreasing the pressure from distal to proximal. The purpose of gradient pressure is to improve venous and lymphatic return, to support arterial supply, but not enhance it, and to assist in hemostasis after wound excision. The entire bulky dressing can be reinforced with a dorsal or a palmar plaster or plastic orthosis, or both. The plaster should not contact the skin. A single layer of Webril (Kendall Healthcare Products Co.) interface is needed to ease plaster removal. The orthosis can be secured with an elastic wrap or Kerlix (Kendall Healthcare Products Co.) wrap.

When thermoplastic immobilizing orthoses are used, they may be fitted over a single layer of nonadherent gauze, over a bulky wrap, over a stent dressing, or over a wet fine-mesh gauze dressing. Postoperative skin grafting orthoses may be fabricated preoperatively, intraoperatively, or postoperatively. Orthotics applied in the operating room fit well initially since they are molded to the final dressings after grafting. The area immobilized includes the grafted area and the immediate joints proximal and distal to it. Any plastic orthosis can be cut open so drainage can escape. Very wet dressings if not changed frequently, can cause maceration and superficial infection.

When the anesthetized casualty is fitted with an orthosis, it is important for the body parts to be aligned in an anatomically correct position. Casualties adjust easily to wearing an immobilizing orthosis when adequate pain medication is administered postoperatively. Later, when pain medications are being decreased, the orthosis often feels comfortable, which increases wearer acceptance.

Areas That May Require Immobilization Orthosis

Face and neck. Halo skeletal traction is sometimes used. Thermoplastic halo immobilizing devices are difficult to fabricate and allow some movement, but are sometimes used. The halo with a thermoplastic jacket with a very high trim line, which extends onto the neck, totally immobilizes the neck.

Shoulder. Skeletal traction or foam wedge pillows optimally immobilize and position the shoulder. These methods are most commonly used to manage axillary immobilization. Rarely, when total contact or 3-point airplane splints (see Figures 10-56 and 10-58) are fitted postoperatively, they position the shoulder in up to 120° of abduction and 10° to 15° of shoulder flexion to prevent a neuropathy.[67] If needed, a separate orthosis supports the elbow in slight flexion and the wrist in slight extension. A total contact airplane splint, formed over the under surface of the abducted arm, keeps the graft from shrinking and distributes the pressure over a grafted area (see Figure 10-57) more accurately than the 3-point airplane splint. Prosthetic elastomer inserts may be molded to the axillary area before the splint is fabricated to increase the conformability of the splint to the axilla. The 3-point airplane splint does not immobilize as well and allows problematic webbing (shrinking of the anterior and posterior axillary fold skin limiting shoulder movement) between the chest wall and the arm[65] (see Figure 10-58).

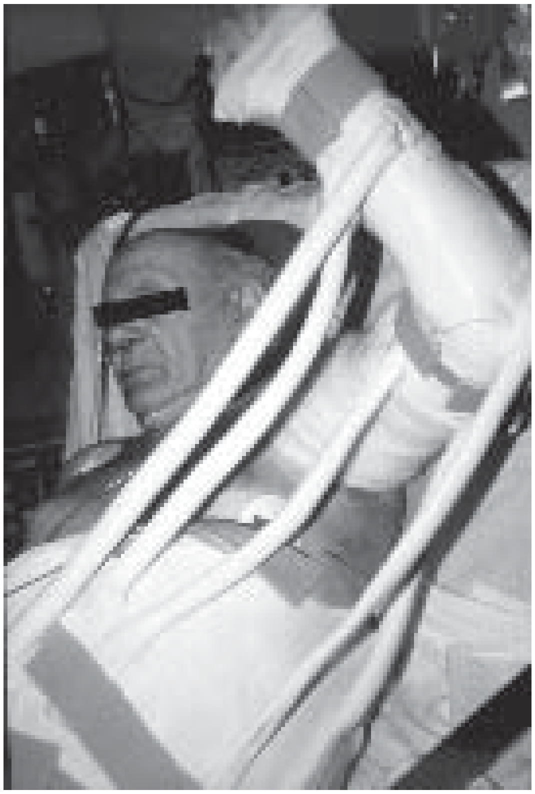


Fig. 10-58. Airplane splint.

Elbow. A volar extension splint or posterior flexion splint may be made to keep an elbow graft in place.

Hand. When a pressure wrap is not used for postoperative hemostasis, a WHFO (see Figure 10-40) is fabricated to keep the IPs extended, the MCPs flexed at 65°, the wrist extended to 20°, and the thumb abducted. It is important to include the palmar arch contour in the orthosis since the arch is necessary for opposition.

Hip-knee-foot. The hip, knee, and foot positions can be immobilized with a commercially available foam AFO with a derotation outrigger. A bulky pressure wrap with cotton batting, followed by gradient pressure elastic wrap is used, and the leg is positioned with the knee above the heart and the foot above the knee for control of postoperative bleeding. An OCL plaster support for 90° ankle may be fitted. When only the ankle needs postoperative positioning a posterior thermoplastic or plaster ankle-foot-orthosis may be used.

Functional Activities and Adaptive Equipment Use During Immobilization Phase

In the immobilization phase it is important to avoid trauma or shearing the skin surface during ADL. Performance of functional activities such as feeding (Figure 10-59), grooming, and communication are appropriate as long as the graft is immobilized. Adaptive equipment is continued as needed. Applying lotion to healed or unburned areas increases sensory input and decreases hallucinations from sensory deprivation. Additional functional activities that can be performed in the immobilization phase are sedentary recreational activities such as viewing television or the VCR. It can also be therapeutic to watch educational videos regarding burn treatments. In addition, the casualty can participate in activities with others, such as board games, cards, or crafts if the graft is protected.

Fig. 10-59. Feeding during the immobilization phase.

Psychosocial Adjustment During the Immobilization Phase

The immobilization phase following skin grafting may be very difficult. Preparation for grafting includes briefing the casualty and family about the procedures and time frames of the immobilization. An informed soldier will experience less unfocused apprehension concerning the operation. Increased acceptance of the procedures is observed when both the family and soldier understand the need for grafting and the alternatives for achieving wound healing. The surgeon and rehabilitation team reassure the soldier by helping him redefine the grafting operation in a positive light, rather than as a setback, and allay fears related to loss of control during anesthesia.

The casualty is not able to exercise the grafted area or perform as many activities due to potential skin shearing from movement. The resultant lack of movement and sensory deprivation create the potential for confusion and hallucinations. The potential for confusion or hallucinations is reduced by staff or family providing appropriate sensory stimulation, especially if the casualty is sensory-deprived on a Rotobed or a Clinitron bed.[70] The use of tactile input through rubbing lotion on closed areas will provide itch control as well as a sense of reassurance through therapeutic touch. Auditory and visual input may be emphasized through the use of television, radio, music, and frequent family and staff interaction.

Explanations of procedures before they are performed provide reassurance. Adequate analgesia and sedation as well as the use of familiar relaxation exercises help to provide a level of comfort that is tolerable during this phase. Avoiding repeated, unexpected, painful, and frightening treatments reduces the potential for developing posttraumatic stress disorder (PTSD). The staff should provide patient choices as much as possible. The soldier should be invited to participate in procedures such as staple removal, wound cleansing, and antigravity positioning, thus reinforcing the casualty's control and responsibility in the healing process.

Wound Maturation Phase of Healing

The goals during the wound maturation phase of healing are to:

- assist in nonnarcotic analgesia;
- maintain wound closure;
- prevent infection;
- control edema;

- regain joint and skin mobility;
- regain strength and endurance;
- facilitate casualty and family participation in resumption of family roles;
- assist learning self-care;
- fit total-contact, stretching orthoses to minimize hypertrophic scar formation;
- learn compensation techniques for exposure to friction, trauma, ultraviolet light, chemical irritants, extremes of weather or temperature;
- develop awareness of sensory changes;
- fit prostheses;
- develop a profile for active-duty training, return to part-time modified duty, or to full-time active duty; and
- continue counseling to deal with life and psychological stresses regarding permanently changed appearance, altered ability levels, and difficulties with posttraumatic stress symptoms.

Casualties with large, deep burns may not become independent without being transferred to a rehabilitation center. For transfer, the casualty should be medically stable and able to tolerate and benefit from a minimum of 3 hours a day of therapy. Appropriate general goals for the burned soldier treated at a rehabilitation center include the following. The patient will:

- be able to direct skin and wound cares, need minimal assistance with bath, and be improving in self-care and independent donning of external vascular supports;
- recover preburn AROM and be able to demonstrate prolonged stretching to decrease joint and skin contractures;
- recover preburn strength as compared with normal for his age group;
- develop endurance to tolerate 2 hours of work equivalent activity and 10 hours of activities, including homemaking or home chores;
- recover coordination sufficient for work and to accomplish daily living skills with minimal adaptive equipment at preburn speed;
- control limb edema using external vascular supports 23 hours a day and elevated positioning;
- demonstrate independent donning of orthoses to modify burn scars, wear devices the prescribed number of hours, and explain the purpose of these devices;
- learn and use protective interventions for sensory, vascular, and pigment changes;
- have healed burn wounds, grafts, and donor areas and will demonstrate care of blisters;
- demonstrate successful use of interventions to control itching;
- participate in appropriate, coordinated discharge planning;
- participate in family education;
- explore vocational issues with a vocational rehabilitation counselor;
- explore and participate in recreational activities, leisure planning, and social and community reintegration while wearing external vascular supports and splints;
- demonstrate consistent independence in self-care and homemaking; and
- be learning to cope constructively with posttraumatic stress symptoms, changed body appearance, and adjustment to disability.

Wound and Skin Care and Nursing During the Wound Maturation Phase

During the maturation phase of healing the epithelium in the healing partial thickness burns is very fragile, and if not protected, is prone to shearing, pressure, and subsequent breakdown. It is not uncommon to have small blisters form during this time because the epidermal layer is not firmly attached to the underlying dermis for several months.[56] All areas should be gently cleansed and rinsed well with water. Infection is no longer a consideration, and therefore, antibiotic ointments are discontinued. Blistered areas should have light, nonadherent dressings applied for protection. The intact blister should not be opened or debrided. If the blister is large and spreads when the external vascular support wrap is applied, the blister should be drained and the wound protected with the dry skin. If the dry skin peels away, the open area is treated with Unna ([ConvaTec] ER Squibb & Sons) dressing, Merthiolate ([thimerosol] Eli Lilly & Co.) to dry the area, or other topical medication if infected. Assessment is made for applying external vascular support to protect the skin and decrease edema. Use of external vascular support garments is begun when open areas are dry, will not adhere to the garment, or tear with garment removal. Careful inspection of the casualty's skin should be ongoing for any condition changes resulting from increased activity, exercise, and the skin's response to treatment procedures. Figure-8 elastic wrapping should be applied to the lower extremities prior to dependent positioning or ambulation.[106] Standing in the shower with legs dependent is permitted only after all open areas are closed on the lower extremities, and purple color of dependent wounds is decreasing.

After a burn injury the number of sebaceous and apocrine glands are decreased, therefore, the healed skin may appear dry and flaky. The casualty may be very distressed by pruritus. Vigorous rubbing or scratching results in newly opened areas. Moisturizing lotions should be applied to all healed areas after bathing and routinely as needed. The lotion should not be perfumed, have an alcohol base, or be so viscous that it causes blisters during application. Adequate dosages of long-acting oral antipruritic medications should be used[107] in conjunction with lotion to modify the itch. Fingernails should be kept trimmed, smooth, and clean to prevent excoriation of fragile skin. Desensitization exercises and vibrator use (Figure 10-60) may be helpful to reduce itching. As sweat glands, sebaceous glands, and hair follicles return, the casualty may notice a condition similar to acne caused by plugging of pores. A grit soap and cleansing sponge such as Buf-N-Puff (3M Healthcare) may soften the tough outer layer of keratin and allow pores to function normally. When apocrine function returns, it is not uncommon to have excessive sebum production and perspiration. The casualty then benefits from a more drying, alcohol based lotion. If sunscreen protection lotions are being used, it is important to prescribe the appropriate type of sunscreen according to the natural condition of the skin. The avoidance of sun exposure for about 1 year is the best method of preventing permanent hyperpigmentation (see Figure 10-32).

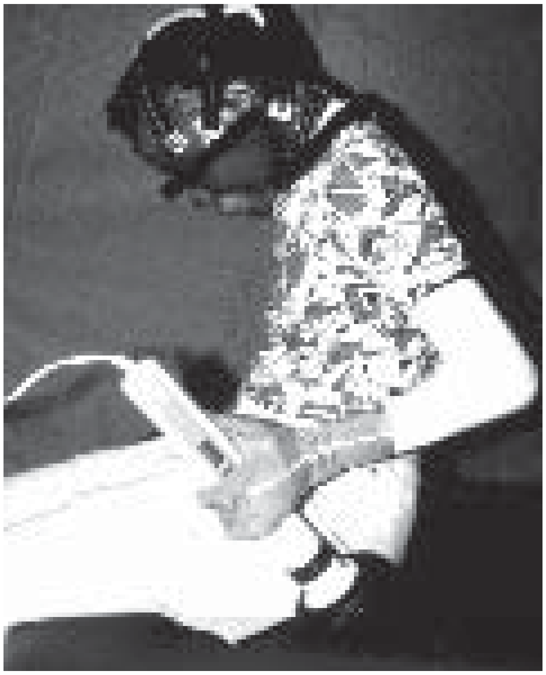


Fig. 10-60. The casualty is desensitizing the scar area using a vibrator.

During this maturation phase the burn casualty must come to terms with being a survivor and prepare to leave the safe environment of the burn unit and reenter society. This reentry may be done in steps: an excursion outside the burn unit, a day pass with family or friends, passes to restaurants, or trips home. These outings will help the soldier to adjust to stares and rude comments. It can also help problem solve for the discharge date as to what adaptive equipment will be needed at home. At this stage, these casualties can benefit from talking to other burn survivors, a psychologist, psychiatrist, or social worker, and from viewing videotapes about other burn survivors and their families to learn how they coped and what their questions and concerns were. Along with physical and emotional recovery, ongoing social support is a necessity for the burn survivor to be a "survivor" and not just another "victim." Nurses provide psychological support and encouragement to the burn casualty as he prepares to leave acute care and return to society. At this time nurses also recommend referral to appropriate services such as alcohol and drug treatment facilities or chronic pain programs. Studies[108] have confirmed that burn victims who are likely to abuse drugs or alcohol or both did so before the accident. Outpatient treatment in these situations may be indicated.

Although no direct central nervous system damage occurs from a burn, most burn survivors notice changed reflexes and changes in their perception of tactile and visual information. Proprioception is often diminished. The itching, tenderness, and skin pain subside gradually. Before the soldier returns to coordinated independent activities that could be dangerous, such as getting into and out of the bathtub alone, driving, using power tools, climbing ladders, or cooking, he should practice these activities with supervision. Medications that cause drowsiness should be discontinued prior to the operation of power equipment or motor vehicles.

A desensitization program will allow the soldier to control the level, frequency, duration, and pressure of differing external stimuli applied to a healed wound. As the casualty's tolerance increases, his ability to withstand unexpected stimuli improves.

Sensations such as pain and itching from the healed wound decrease very slowly. Sensations from the healed wound become closer to the preinjury feeling more rapidly when a desensitization program including massage, vibration, and exposure to varying textures and temperatures is practiced regularly (see Figure 10-60).

Massage desensitizes the skin and assists venous return. It is manipulation performed with the hands to produce effects on the neuromuscular and circulatory systems. The direction of the massage should be with the venous and lymphatic circulation. The purpose of massage is to relax as well as to improve circulation from the limb. Using too much pressure causes pain or blisters. The massage should be a slow repetitive motion, moving distal to proximal. The limb should be elevated above the heart during massage.

Another method of desensitization involves using wooden dowels, wrapped in various pieces of cloth, to produce repeated stimuli over the affected area, which increases tolerance. Desensitization can be performed by graded contact with particles that produce different sensations. The hand or foot may be immersed in a basin that contains various objects such as cotton, popcorn, plastic insulation pieces, rice, plastic squares, or beans.

Vibration is a controlled method of stimulation of nerve endings that may lessen pain, response, or irritation. An electric or battery operated vibrator is used to massage around the areas surrounding a burn. As tolerance increases the vibrator can be placed directly over the healed wound, first with a stocking covering the skin and then directly against the healed skin.

Another desensitization method is controlled exposure to warm and cool water. The extremity is maintained at heart level, and water is allowed to run over the healed wound: begin by running the cool water over the wound for 20 seconds, followed by warm water for 5 seconds, and repeat this procedure for up to 10 minutes. Contrast baths of warm, followed by cool water may be substituted by dipping hands slowly into the contrasting water.

Burn recovery, return to active duty, and return to a normal home life usually include resuming sexual relations.[109] Former burn patients report that regaining their sexual identity is their most important goal for recovery. They have been suddenly and traumatically shocked into realizing their vulnerability. They want to deepen and strengthen relationships with a domestic partner and are afraid of rejection. They may seek satisfying, relaxing intimacy even if that was not important to them before the accident. They find it reassuring when their doctor initiates a matter-of-fact discussion of sex and birth control in the same way he discusses other medical aspects of their care.

The soldier's partner needs to be included in discussions about changes that take place during recovery and the expected final outcome. The casualty must take responsibility to initiate communication with his partner, and must understand that human sexuality is communicated to the partner through behavior, appearance, and personal hygiene, which reflect the soldier's self-image. Most companions appreciate an open discussion prior to resuming sexual activity.

The casualty often worries about rejection. Counseling to encourage trusting, honest communication can become the basis of a loving, caring, exciting relationship. Understanding, communication, imagination, and experimentation can expand opportunities for a good relationship.[110] Potential problems related to sexual activity as well as birth control should be addressed by the physician before the soldier is discharged. Friction or trauma to healed burns during sexual activity can cause blisters. These blisters may heal slowly. They are normal and decrease as skin toughens and matures. A lubricant decreases friction. The areas least burned are helpful in sexual activity. A commonly used means of sexual expression is the mouth. The casualty may receive more pleasure from this option, because the lips and tongue are rarely severely burned and are more sensitive to touch and temperature than other body parts. Any part of the body can be made clean enough for oral contact.

Pregnancy may cause hypertrophy of burn scars and should be avoided until the scars are mature. Burns of the female genitalia are very rare because these structures are unusually well protected. However, contractures with shortening of the skin around the inner thighs and lower abdomen can be a problem. Burns of the male genitalia often need urologic consultation to achieve optimal outcome. Stretching the tissue away from the direction of contracture and frequent erections assist wound maturity. Vigorous exercise and stretching of these areas as healing progresses will result in the best outcome. Contracture releases are possible.

Talking together, kissing, and caressing each other in a tender, sensuous way assists arousal, which is commonly more difficult after the burn injury. It is common for the casualty to be anxious. A major part of sexuality depends on the way an individual chooses to use the body, attitudes about a variety of sexual behaviors, and informed choices made based on internal feelings of competence, effectiveness, and self-satisfaction. Recovering sexuality and intimacy after a burn injury is a dynamic process that can sometimes be facilitated by a counselor or trusted confidant.

Erickson's[111] sexual counseling approach was to avoid pointing out or interpreting patient's fears. He did not emphasize insight. His approach was based on action to bring about change and he emphasized the positive. His focus was on expanding the person's world, not educating him about his inadequacies. When working with couples, Erickson often relabeled what they were doing in a positive way. He attempted to gain a small response and build on that. In this way, positive forces were freed to allow the couple's further development. Couples who had worked out an amiable way of living together before a stressful event, such as a burn, with positive support and commitment to each other, resolved sexual difficulties. One of Erickson's basic premises was that the art of marriage included achieving independence while simultaneously remaining emotionally involved with one's relatives. This type of adjustment is crucial for the burned individual, whose full recovery is dependent on his ability to capitalize on family resources.[111]

Exercise During the Wound Maturation Phase

Full AROM returns most quickly when inflammation is minimal, when grafts are on dermal remnants, and when the casualty continues hourly, elevated, active motion during waking hours. The burn casualty needs to exercise for strengthening (AROM against gravity will increase strength when less than antigravity strength is present), for improving speed and accuracy of movements, for flexibility, and to improve skill in activities. With prompting, the casualty who has had adequate analgesia during the first two phases will put out maximum effort to move through the extremes of motion. This will prevent joint motion from becoming painful, nourish joint cartilage, and elongate surrounding soft tissue. Exercise continues to speed healing by improving circulation and by decreasing edema and the inflammatory response to burn injury. At this phase of recovery, the casualty should exercise wearing external vascular supports. It may be necessary to remove the support garment for 10-minute periods for composite stretching, but the support must be donned immediately after the exercise. The garment can only be removed at the distal limb. If the garment must be off an elbow, the whole sleeve must be off, or an elbow zipper can be opened for prolonged flexion and supination but it must be closed after exercise. A glove must not be removed without also removing the sleeve, which presents complications if the sleeve is attached to a vest.

Former patients report that to be effective, exercise periods require mental concentration as well as vigorous physical investment. The casualty should eliminate distractions such as visitors during this important time and concentrate on restoring greater function with each exercise. A clock that announces time intervals, such as ringing every 15 minutes, assists the soldier to remember to stretch eyelids or other important contractures throughout the day without the verbal cues that may come to seem like nagging from a therapist.

Manual resistive exercises, progressive resistive exercises, use of Cybex (Cybex Co.), BTE (Baltimore Therapeutic Equipment), rowing machines, stair climbers, bicycle riding, and other therapy modalities should be done daily to improve strength and endurance (see Figures 10-32 and 10-61). It is generally considered that high repetition, low resistance exercise increases endurance, while lower repetition, high resistance exercise increases strength. However, strength can be increased with low

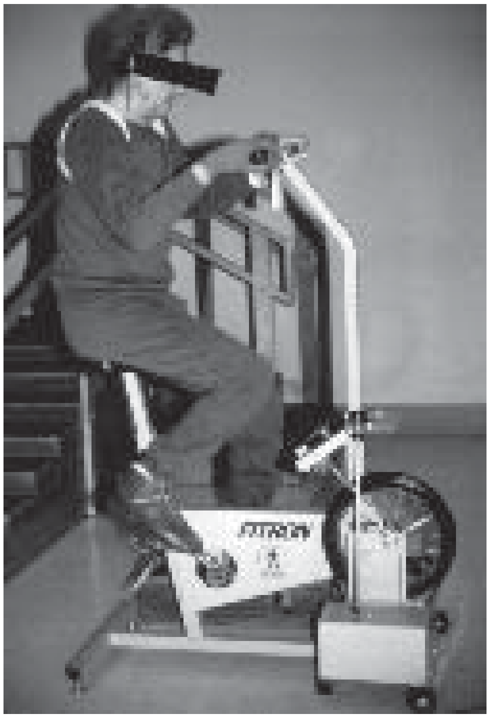


Fig. 10-61. The stationary bicycle provides strengthening, active range-of-motion, and endurance exercise.

weights as long as fatigue occurs during exercise. Walking continues to be a favorite aerobic exercise. Furthermore, walking restores good posture, ie, body straight, chest up, chin in, shoulders back and down, and hips tucked in. When ambulation is done out of doors, it provides distraction from pain with focus on the surrounding scenery.

During this phase of wound maturation, prolonged stretching is almost always recommended for decreasing joint contractures. Elbows remain susceptible to heterotopic ossification when they are stretched too vigorously. However, gentle, graded prolonged stretch, preferably active, will assist the collagen to realign in a longer, less tightly convoluted, mat-like configuration. Contractures at this point are usually so severe that they will not resolve without prolonged stretch in addition to the strengthening exercises. Adequate time for prolonged stretch and composite motions must be reserved by the therapist because the patient will almost always need coaching and hands on supervision for this part of rehabilitation. The other modalities can often be incorporated into written, home programs and sometimes can accompany prolonged stretch. Stretch can be combined with work-equivalent activity. Sometimes an activity such as having the casualty copy an enticing advertisement with one hand, while the therapist stretches the other hand, helps the soldier to relax and benefit more from the stretch.

Varying the exercise program makes it more enjoyable. Individual sports such as biking, jogging, and tennis are wonderful beginning exercise. Graded to increase as endurance increases, these sports return control to the soldier. Caring for pets is excellent exercise and increases variety and pleasure. If the animal lives in a barn, open areas of the casualty should be healed or well covered to avoid infection. Exercising by doing activities that were routine and important to the soldier before the injury rewards performance (Figures 10-62 and 10-63). When the person is discharged from the hospital, doing the motions of active duty, a job, homemaking or similar activity strengthens muscles. This focuses attention on habitual activities and toward the outcome or product of work. Success is motivating even when it takes longer to do a task after burn recovery than it took before the injury.

Outpatient treatments have the additional benefit of establishing a pattern of leaving the protective home environment every day. Objectively documented improvement is encouraging. Attending a health club increases social contacts and may improve self-esteem as strength and endurance increase. Swimming is an exercise recommended by former burn patients. Swimming in chlorine water is irritating to newly healed skin and external vascular support garments. Therefore, after swimming, the chlorine is washed off; lotion is applied; and clean, dry, support garments are donned. During swimming, the benefits of exercise combined with joint protection and skin moisturization outweigh the nuisances of showering, extra laundry, and lotion application.

Fig. 10-62. The casualty is repairing a small engine as part of overall rehabilitation.

It must be remembered that when exercising out of doors, the patient's healed burned skin or donor areas should be protected from sunlight until all the red color has faded. Damaged, thinned skin will turn very dark brown with even brief sun exposure. Healed skin will tolerate graded sun exposure 3 to 18 months following injury. However, exposure to sun must be gradual and a commercially available waterproof sunscreen with a sun protection factor rating of 15 or more should be applied to all burned areas when exposed to the sun. A wide brimmed hat can protect the ears and nose during exercise in the sun. Sunburn through exposed custom fitted elastic external vascular support garments is common.

If the soldier's skin does not sweat, he should avoid prolonged exposure to temperatures above

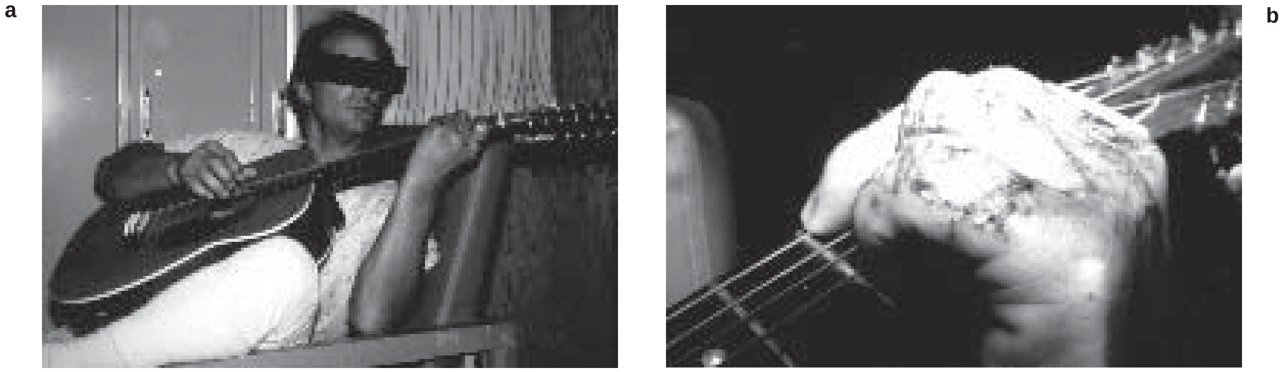


Fig. 10-63. Resumption of recreational activity after skin grafts provides exercise during an activity that was routine and important before the injury. (**a**) Recreational outlet provides therapeutic activity. (**b**) Close-up of grafted fingers exercised into flexion to play the guitar. Skin durability gradually increases during practice.

85°F. A fan or air conditioner may be needed in the summer to aid the body in cooling by evaporation. Hyperthermia during exercise is possible. The casualty must remove garments and orthosis and must shower and cool down after vigorous exercise, then reapply scar management devices. The opposite of hyperthermia and sunburn, frostbite injuries are a risk when the soldier is exercising out of doors and the temperature drops below 32°F. Casualties who have sustained frostbite in the past are at greater risk for frostbite. Warmer clothing, avoiding constricting bands around the extremities, and wearing several layers of clothing during exercise, reduces risks of frostbite.

Casualties often view hospital discharge as a welcome termination of burn care, exercise, and pain. Often casualties eagerly anticipate what they believe is a well-deserved rest. The recalcitrant person who endures exercise and scar control devices at the hospital may abandon custom-made elastic stockings, traction or total contact splints and activity once he feels safe at home. It is the casualty's dream that home will magically bring back the previous physical and emotional status. However, it quickly becomes obvious that achieving adequate epithelial healing for safe home care is only the beginning of rehabilitation and return to active duty.[112] Work hardening programs are appropriate for severely burned casualties (see Figure 10-62). They have the advantage of providing supervised heavy work, with consultants for psychologic assistance to adjusting to changes and dealing with slow return of endurance and ability. In addition, they make needed adaptations for exercise and work apparent. Work hardening also reinforces protective measures for friction, trauma, chemical irritants, and extremes of weather or temperature.

The best outcomes result when the casualty, therapist, and physician formulate an appropriate active duty limitation outline with each clinic visit. Returning part time, in whatever limited way is necessary, to as many active duty tasks as possible is also excellent exercise. Jobs done by habit eliminate the need for the soldier to constantly think about the discomfort of exercising.

Positioning in the Wound Maturation Phase

The goals and techniques for positioning during the wound maturation phase of healing are very similar to the acute phase already discussed (see Figures 10-30 and 10-34). Initially, dependent positioning of the extremities will be painful, which will remind the soldier to elevate hands or feet. Later, as the soldier must take responsibility for varying his own position and using antigravity edema management techniques, a kitchen timer ringing every hour is a nonthreatening reminder.

Water beds are contraindicated initially for sleeping due to the rounding of the neck and shoulders and difficulty getting out of bed. In addition, if kept warm, they increase perspiring and itching. Antideformity and antigravity positioning is continued for at least 4 to 6 months postdischarge from the hospital.

Orthosis in the Wound Maturation Phase

Especially during the first 4 to 6 months after the wound is closed, the use of orthotics does not replace exercise. Orthotics (in addition to the functions discussed in the previous sections) stretch contractures, substitute for lost function, and minimize scars.[66] Individualized, custom-made orthotics enhance positioning, allow reasonably painless maintenance of exercise gains, block undesirable motion, and encourage active motion away from a contracture. Custom made orthotics assist in minimizing hypertrophic scars by flattening the hypertrophic tissue against the underlying body structures. Serial drop-out casts, used for progressive scar elongation, have the additional advantage of softening the scar, preventing orthotic slippage, and eliminating patient removal.

Head and neck. During the maturation phase, the preservation of facial contours, while applying pressure to reduce hypertrophic scarring is an ongoing challenge. The position of teeth and the facial contours are developed from the counter pressures of the tongue against the teeth and the active motion and tension of the external facial musculature. The presence of a contracting scar or the use of an orthosis will alter the balance of these mechanics. A dental consultation is therefore recommended every 4 months for a patient needing external vascular supports for management of facial scars. A transparent facial orthosis or neck orthosis, described later, provides optimal management for face scars or oral contractures. The MPA can continue to be used for oral contractures (see Figure 10-47). The Macfarlane microstomia correction device (Figure 10-64) maintains horizontal oral aperture gains.[113] The Macfarlane device has the additional benefits of preventing tooth migration when it is worn daily and fitting well with a clear orthosis because the portion at the corner of the mouth has no flange. The Macfarlane appliance also spreads the lips over the teeth, so it is easier to blanch the tissue. Unless the dentist determines that the lower teeth are in a solid arch shape, patients who need pressure for scar modification over the lower lip and chin also need a retainer fitted for the lower teeth to prevent lower tooth migration from this imbalance of pressure.

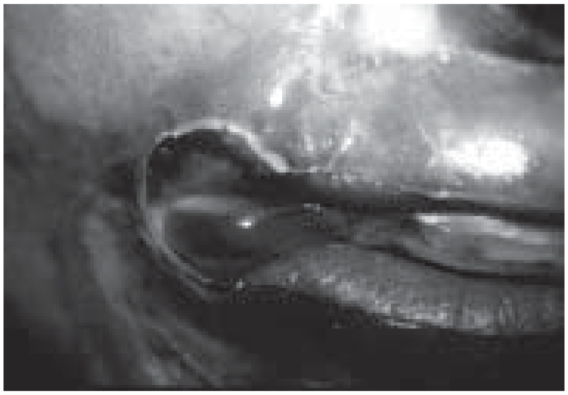


Fig. 10-64. Macfarlane microstomia correction device (shown here) can easily be used with clear facial orthosis.

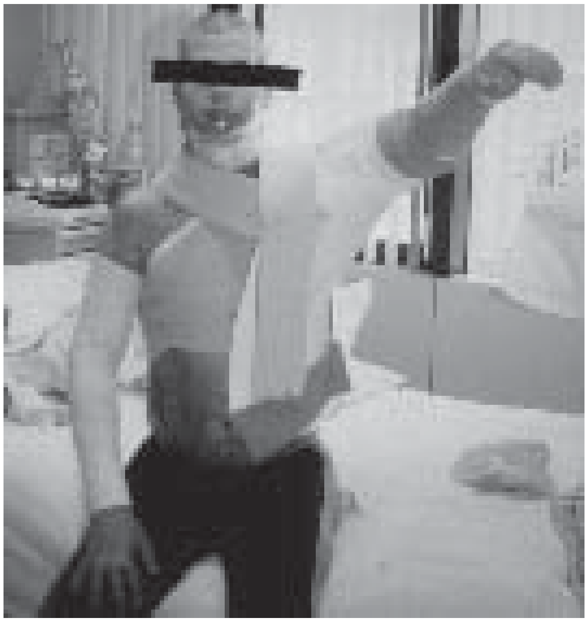


Fig. 10-65. A casualty shown with simultaneously worn chin-neck and axillary-elbow-hand appliances.

The neck responds well to clear cervical collars, but foam or thermoplastic devices also manage scars and prepare the epithelium for more accurate total contact supports. Tubular ring collars can toughen neck tissue and if applied over a smooth contact layer such as Xeroform (Chesebrough Ponds, Inc.), assist in neck scar maturation. Often the chin, neck, elbow, and shoulder need splints at the same time. Making sure they fit together and are comfortable helps the soldier use them regularly (Figure 10-65).

Shoulder. Shoulders may require prolonged attention during the maturation phase. Positioning orthoses, figure-8 straps, deltoid aids, and CPM devices have their place in the treatment of difficult cases. Positioning orthoses, such as the open axillary abduction splint (often called an airplane splint), are seldom left in place for prolonged periods but must be used in conjunction with active exercise. However, total contact axillary orthoses are worn for prolonged times and provide continuous pressure and stretching to hypertrophic webbing bands of the anterior and posterior axillae. When there is early hypertrophic scarring along the anterior and posterior axillary folds and the skin is healed and durable, a commercially available figure-8 clavicle strap may be applied (Figure 10-66). If the tissue breaks down, the strap must be dis-

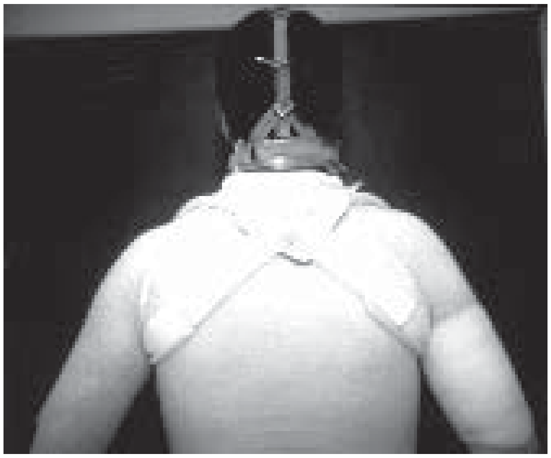


Fig. 10-66. Figure-8 clavicle strap.

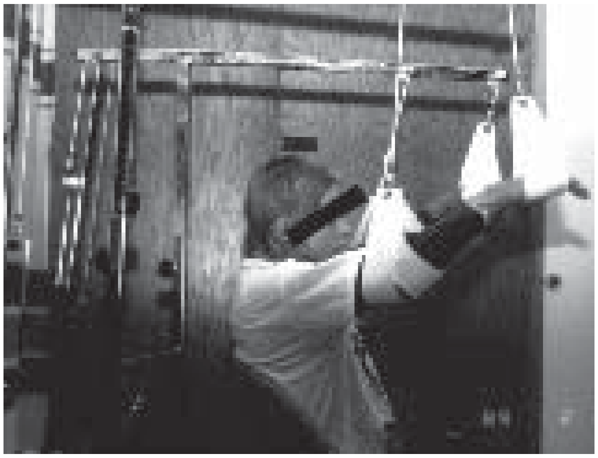


Fig. 10-68. Deltoid aid used as positioning dynamic orthosis.

continued (Figure 10-67). The strap may be worn at night when motion does not cause blistering or irritation if wearing is tolerated and the scar heals. Deltoid aids are utilized to help position for prolonged stretching of contracture bands when the soldier relaxes, and yet allow active motion as well as permit the person to participate in some ADLs (Figure 10-68). CPM devices are helpful to move the shoulder continuously and provide stretching at the ends of motion.

Elbow. The elbow contracture responds best to a the serially revised or "drop out" orthotic (see Figure 10-33). For serial casting or splinting, the device is usually changed every 2 to 3 days to accommodate a resolving contracture.[68] Bivalved casts are difficult to put on, but if the casualty is independent in using them, a prolonged stretch can be achieved when the casualty is not exercising or involved in functional activities. Circumferential bivalved arm casts are applied to achieve independent composite stretching exercises of the shoulder, elbow, and wrist. An elastic wrap can secure these for night wear (Figure 10-69). Elbow CPMs are also helpful.

Hand. The purpose of hand orthoses in this phase is to reduce or prevent deformities or replace lost function. Deformities such as boutonniere (see Figure 10-43) and swan neck are tendon injuries, which need a combined approach by the reconstructive surgeon and the therapists. However, traditional orthotic management (such as three-point flexion or extension splints, extension troughs, and LMB

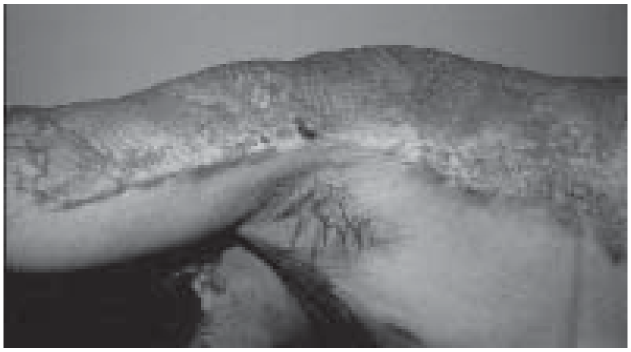


Fig. 10-67. Irritated area resulting in temporary discontinuance of garment. Open area along left axillary contracture, stretching during exercise. Patient discontinued figure-8 clavicle strap during exercise, but wears it at night without increased irritation.

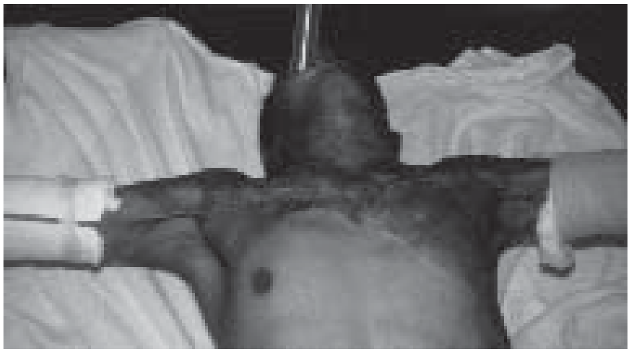


Fig. 10-69. Circumferential bivalved casts assist shoulder abduction with composite chest, and bilateral shoulder and elbow stretching. In this simulation, the patient is simultaneously doing neck extension and mouth stretch.

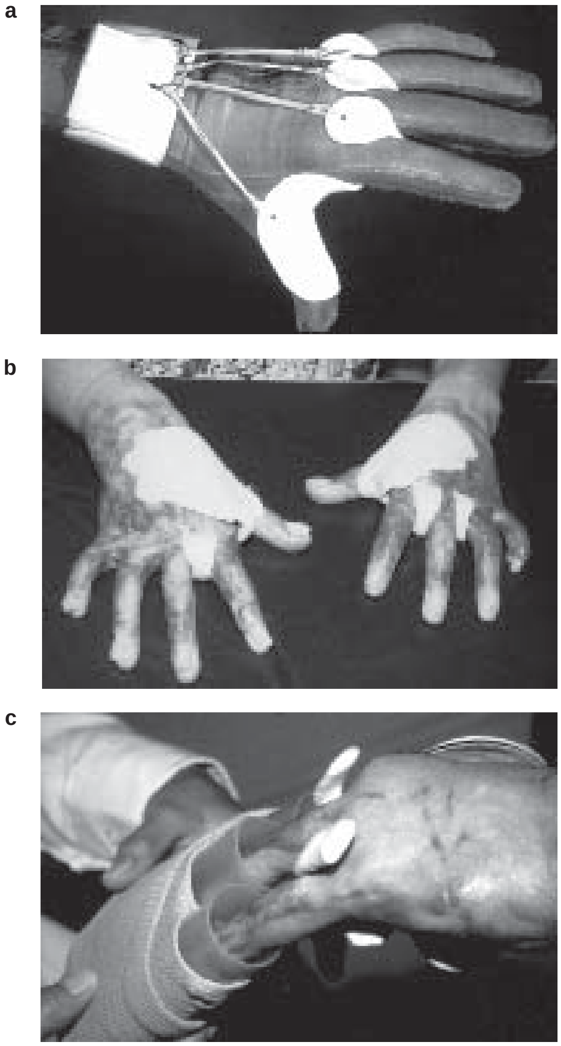


Fig. 10-70. Management of interdigital contractures. (**a**) Thermoplastic interdigital thumb and finger web spacers attached to Velcro wrist band. Wrist band must not restrict venous return. (**b**) Silicone interdigital thumb and finger web spacers prior to glove being applied. (**c**) Felt interdigital web spacers. Note that external vascular support glove is being applied with the aid of tubes.

[North Coast Medical] flexion or extension springs or joint jacks) is used in maturing burns as the healed epithelium becomes tough enough to tolerate these splints. Conforming total contact thermoplastic or silicone orthotics are used in the hand to apply pressure to contracture bands. The interdigital web space and thumb web contractures are especially responsive to silicone inserts (see Figures 10-46 and 10-70). Concave areas of the hand, such as in the palm, are also improved with silicone inserts, especially when the soldier is not using the hands (Figures 10-71 and 10-72). Total contact pressure on the scar tissue softens and elongates this plastic tissue better than three-point splints. Foam sandwich splints may be used to achieve wrist and digit extension and abduction.[114] This orthosis would be secured with an elastic wrap at this state of tissue durability. Air splint devices also reduce edema and stretch the hand or foot.

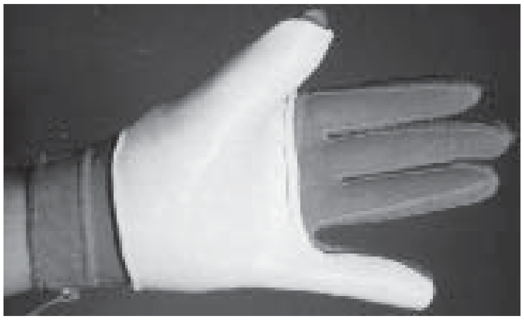


Fig. 10-71. Total contact thermoplastic palm orthosis.

Dynamic splints are not well tolerated by individuals because of discomfort associated with unrelieved, constant stretch (Figure 10-73). Serially revised or drop out orthotics provide slow, intermittent stretching and, therefore, are better tolerated. Serial orthotics may be used for any individual hand joint or for a composite stretch (Figure 10-74). Hand CPMs are also useful for prolonged stretch at the limits of range-of-motion (Figure 10-75).

Orthotics are also fabricated to replace lost function secondary to peripheral nerve injury. Examples are the thumb positioning post used in a median nerve injury or a dynamic extension orthosis for radial nerve injury. When digits have been amputated, temporary prehension posts aid in grasp and pinch (Figure 10-76).

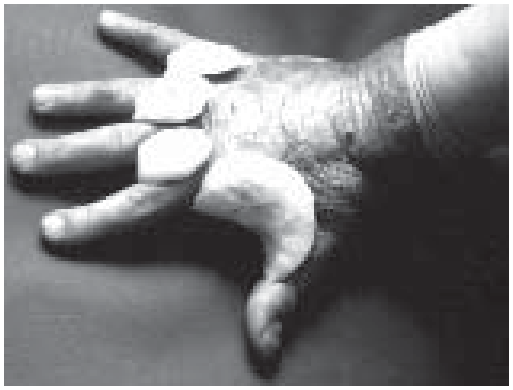


Fig. 10-72. Night interdigital web orthoses. Note elastomer thumb saddle and separate Otoform K finger spacers.

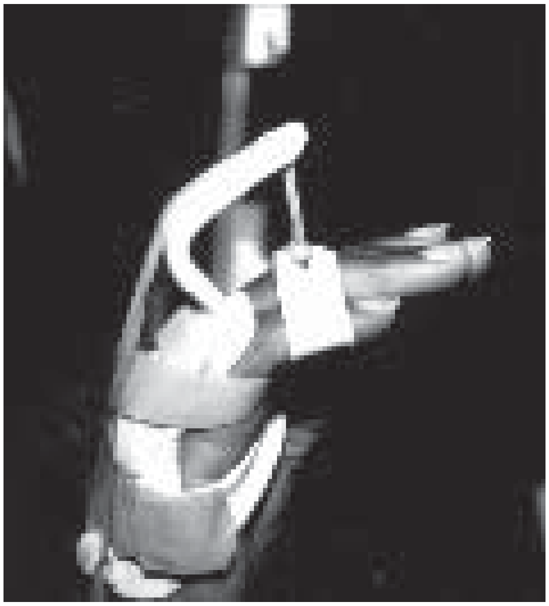


Fig. 10-73. Dynamic elastic thermoplastic extension orthosis for treatment of fifth finger flexion contracture.

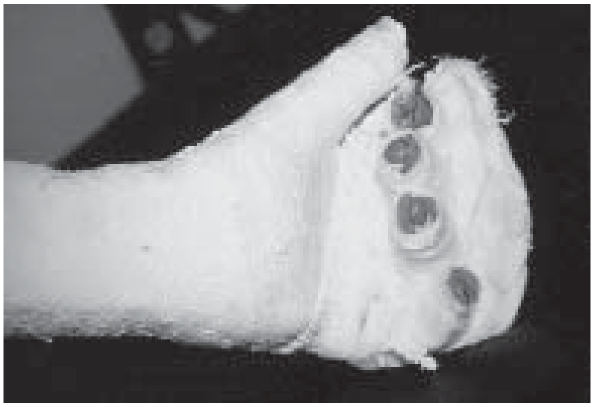


Fig. 10-74. Plaster drop-out hand orthosis.

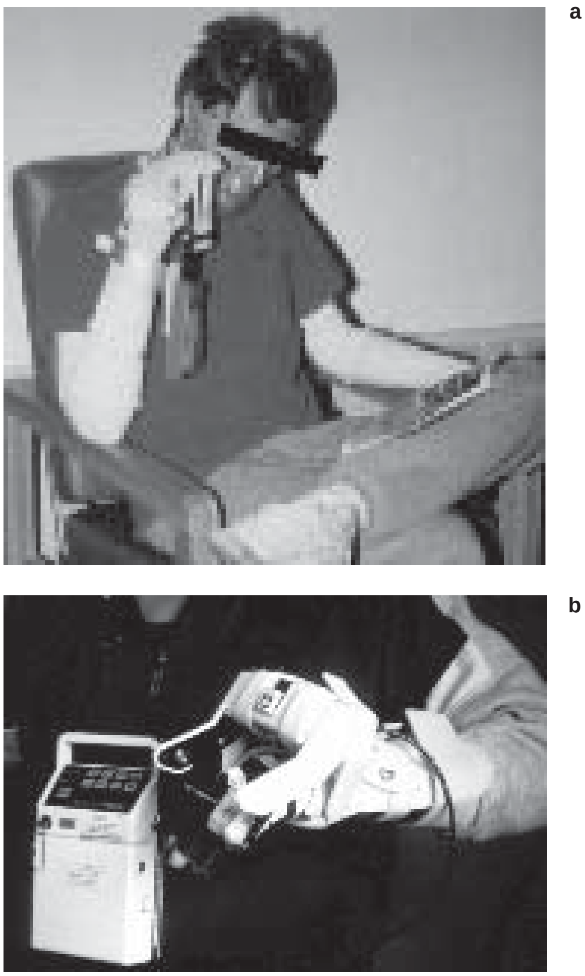


Fig. 10-75. Continuous passive motion machines (CPMs) for the hand. (**a**) Toronto II hand CPM worn as outpatient. Note clear facial orthosis. (**b**) Sutter hand CPM.

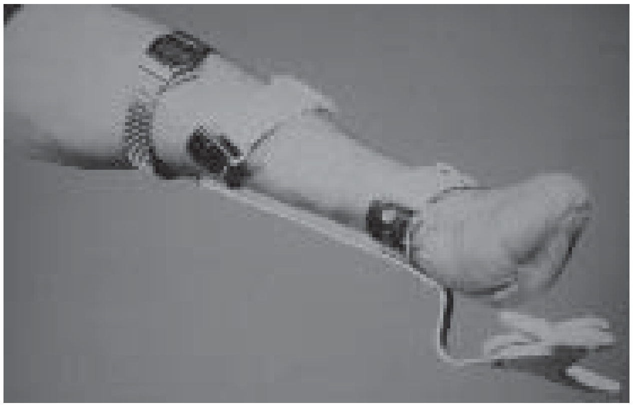


Fig. 10-76. Temporary palmar prehension orthosis.

Knee. A posterior serially revised or drop out orthosis is used at night to stretch resistant flexion contractures of the popliteal space. An orthopedic knee immobilizer orthosis may also be utilized in less severe cases.

Foot. During the maturation phase, foot scars can be very uncomfortable. For people who spend much time standing or walking, a custom shoe and total contact insert may increase comfort by redistribution of pressure. Cutouts such as donuts are never appropriate since they cause edema formation into

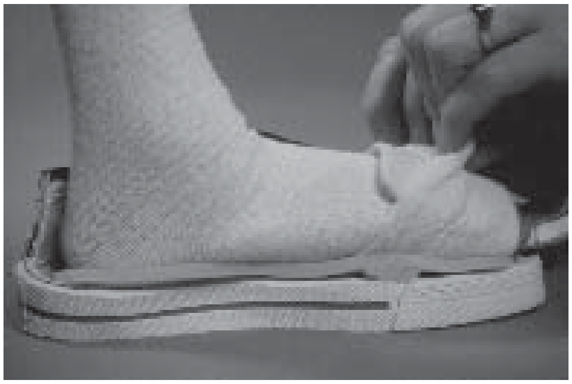


Fig. 10-77. Cloth shoe with custom-fitted total-contact sole insert, with metatarsal bar, and silicone insert over dorsum of toes.

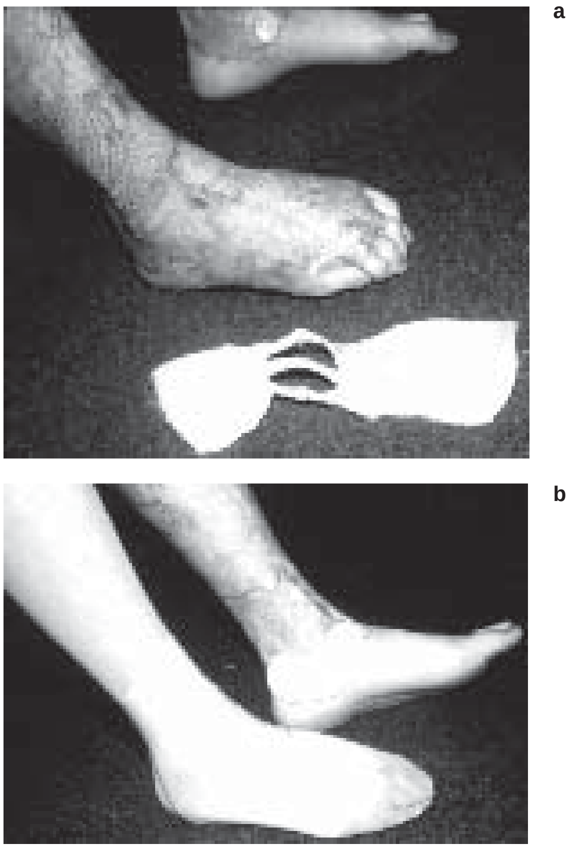


Fig. 10-78. Management of foot scars and contractures. (**a**) Felt interdigital web spacers. (**b**) Elastomer silicone insert below medial malleolus applied under external vascular support stocking.

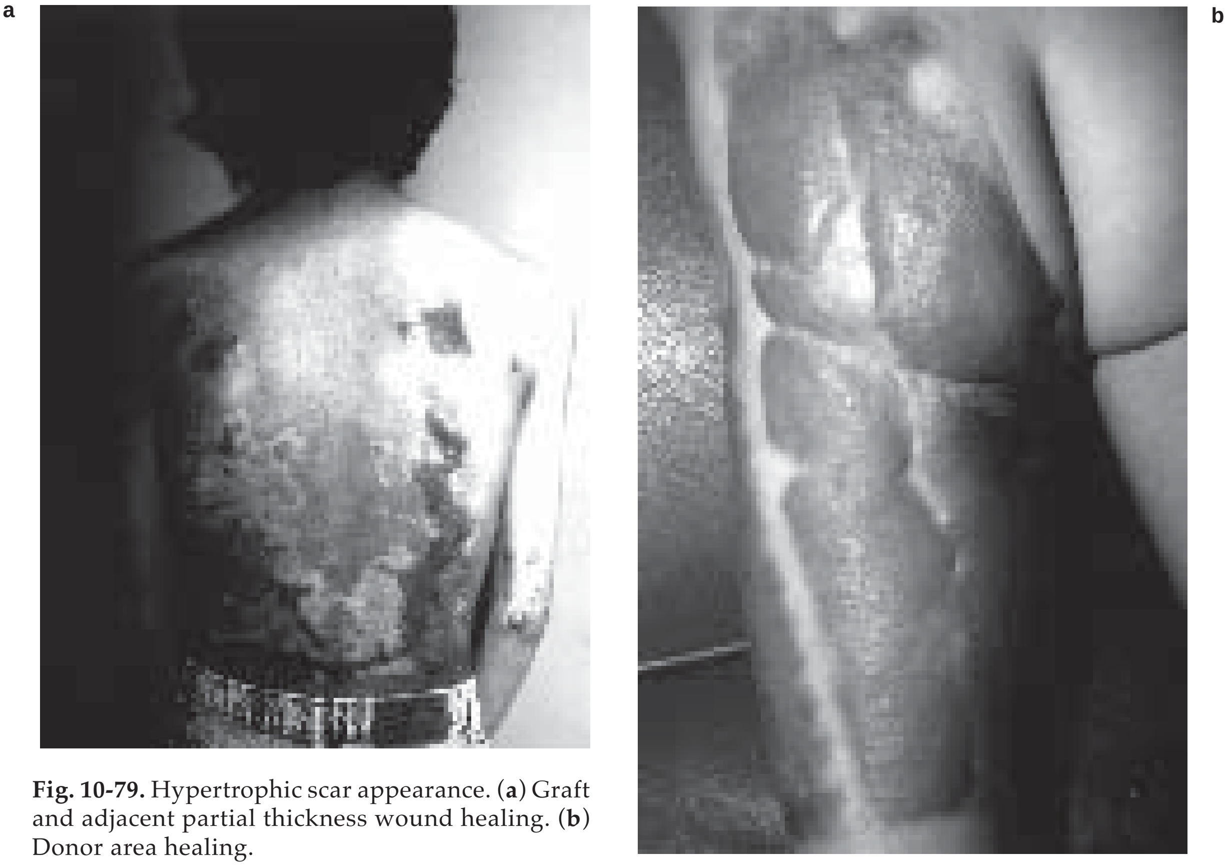


Fig. 10-79. Hypertrophic scar appearance. (**a**) Graft and adjacent partial thickness wound healing. (**b**) Donor area healing.

the relieved area, ultimately increasing scar formation. If forefoot, toe flexion, or extension contractures develop, shoe inserts that apply pressure onto the hypertrophic scar band and stretch the contracture may be required (Figure 10-77). For severe toe extension contractures with hammer toe deformities, a nighttime dynamic toe flexion orthosis will be necessary. A shoe with metatarsal bar, toe insert, and steel shank is another alternative. When toe syndactyl is noted, interdigital web spacers are useful. Elastomer silicone inserts are used to add pressure over hypertrophic scars (Figure 10-78). If a foot drop is present, an AFO may be needed.

Contracture and Hypertrophic Scar Management

The complex pathophysiology of hypertrophic scar formation is not yet fully understood. Unopposed natural wound healing occurs by the processes of contraction in a centripetal fashion and epithelial migration from wound margins and retained epithelial elements at the base of sweat glands or hair follicles.[69] In addition, because wounds into the reticular dermis level are allowed to heal longer than 3 weeks, unnecessarily hard, thick red scars are formed[115] (see Figures 10-67, 10-79, and 10-80).

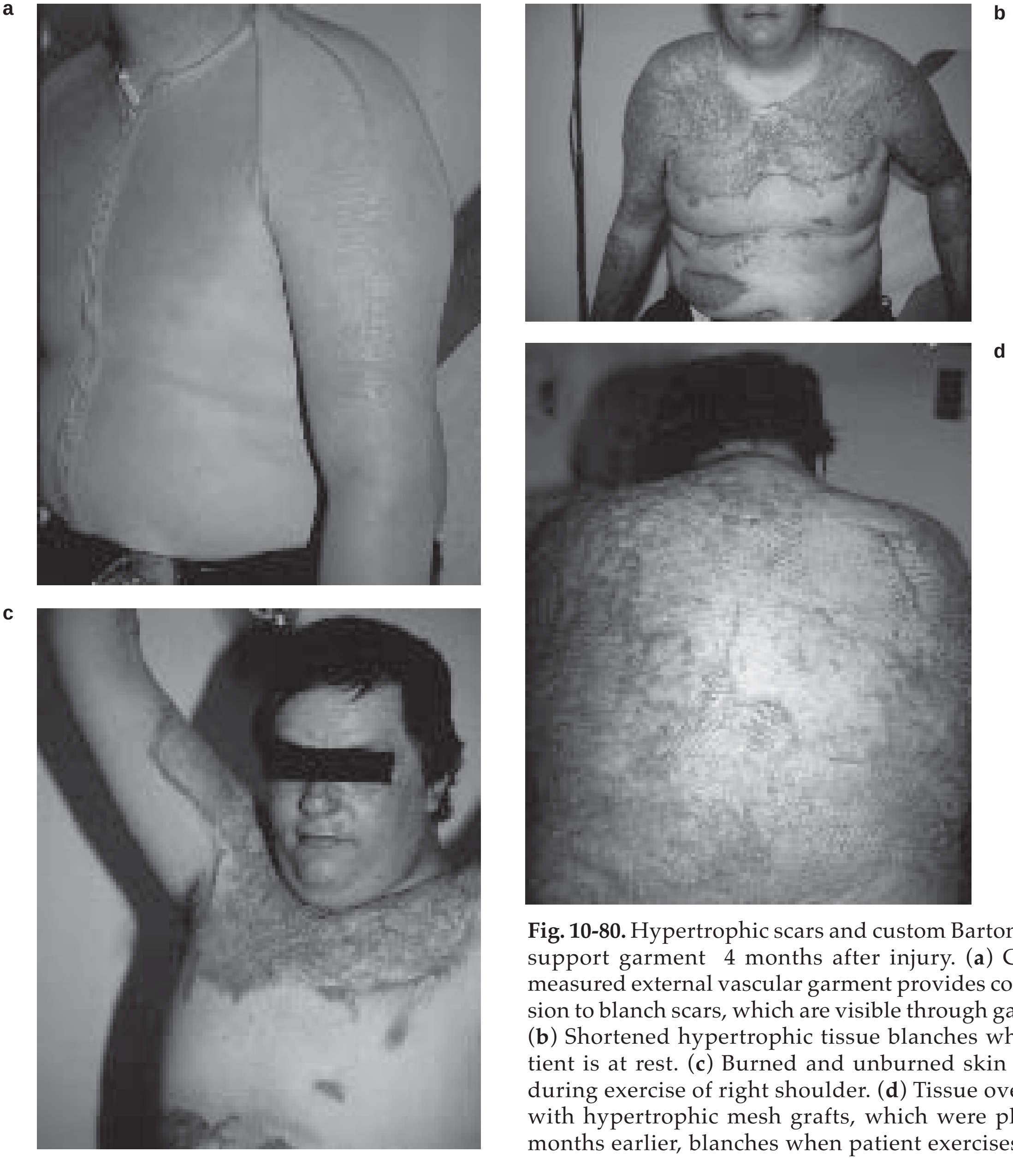


Fig. 10-80. Hypertrophic scars and custom Barton Carey support garment 4 months after injury. (**a**) Custom measured external vascular garment provides compression to blanch scars, which are visible through garment. (**b**) Shortened hypertrophic tissue blanches when patient is at rest. (**c**) Burned and unburned skin blanch during exercise of right shoulder. (**d**) Tissue over back with hypertrophic mesh grafts, which were placed 4 months earlier, blanches when patient exercises.

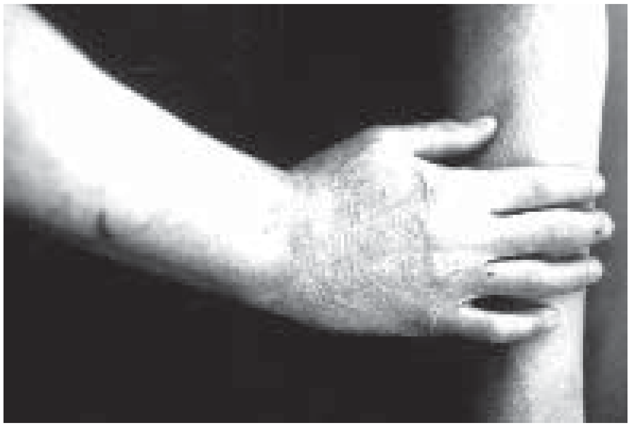


Fig. 10-81. Mature sheet graft over fingers; mesh graft over dorsal hand; and mature, healed, nongrafted forearm.

The duration of the scar process differs depending on the depth of wound and coverage. Full thickness skin graft recipient areas tend to scar less than thin partial thickness grafts. However, full thickness donor areas must be either split thickness skin grafted or primarily closed by undermining the surrounding tissue. The linear scars on tension are likely to become hypertrophic, particularly if they cross perpendicular to the lines of relaxed skin tension. The linear scars may also spread and become unsightly. Skin grafts over granulation tissue or infected granulation tissue scar worse than grafts to wounds excised in 3 days. Meshed grafts leave a permanent pattern of epithelial decussations and interstices. The larger the mesh, the larger the permanent criss-cross pattern. In contrast, a sheet graft, with no hematomas or seromas to disrupt adherence, often appears smooth in 1 or 2 months (Figure 10-81). Donor areas into the reticular dermis are more likely to scar than superficial donors (see Figure 10-79).

During hypertrophic scar formation, there is an increase in collagen and myofibroblast production, a proliferation of new capillaries, with capillary endothelial budding forming a rich vascular network. Fibroblasts appear in the interstitial spaces in greater quantities than normal. These fibroblasts begin to synthesize collagen and produce myofibroblasts that exhibit contracting characteristics similar to a smooth muscle cell. With delayed epithelization, collagen in the connective tissue is produced at 4- to 5-fold the normal rate. The new collagen is profusely vascular, immature, and lacks organization because the interstitial matrices are not correctly regulating its production. The adhesiveness of collagen fibers and bundles, more densely packed and lacking spatial orientation, are associated with a delay in epithelization. Serum protein in the blood inhibits collagenase resulting in a decreased collagen lysis.[116]

The scar tissue begins dynamic remodeling as soon as the inflammatory phase of wound healing is completed. However, the scars are rarely observed with the naked eye until 3 to 6 weeks after the wound is closed. They are noted as hypervascular, dark red, indurated, almost cartilaginous, hard, excessively strong, itching, tender, raised areas at the site of an injury. They may be noted at donor sites if these are taken into the dermis or if the healing donor becomes infected. The myofibroblasts remain active, dynamically remodeling for more than a year. Keloid is often used to designate a larger, thicker scar that grows beyond the limits of the original injured area. The time of scar onset, duration, and regression vary. However, the scar is usually noticed in the first months after healing is complete. If epithelization requires more than 2 to 3 weeks, the scar usually raises above the level of the surrounding tissue. By the third to sixth week, the scar becomes hard, at which time the soldier and physician usually are interested in beginning treatment. The hypertrophic scars spontaneously regress, partially, 1 to 3 years after healing. This regression is age dependent.[117]

The maximum height of scar elevation is usually seen at 3 to 9 months following healing, after which regression begins. Scars will not regress fully if the tissue contours have been altered during the inflammatory phase of healing, when the myofibroblasts were forcefully contracting. Total contact external vascular support by an elastic garment, cast, or plastic orthosis over the healing area reduces circulation, prevents edema, and thereby, interrupts scar buildup.

Factors that influence increased formation of hypertrophic scars include:

- the presence of infection or necrotic material in the wound or both,
- inflammatory response,
- foreign bodies imbedded in the tissue,
- suture materials that act as foreign bodies,
- crushing or irritating the wound during surgical procedures or wound debridement, and
- topical antibiotics that increase the vascularity of the wound.

Another factor that increases hypertrophic scar formation is tension on the healing wound. Tension might result from tight grafts, contractures, mature shortened scar cords in a constantly stressed area such as the anticubital fossa, burns that cross lines of relaxed skin tension, the normal skin elasticity of teenagers, and collagen overgrowth during prolonged debridement and healing. Other influences are a familial predilection or the racial predilection of more darkly pigmented races to form overly protective scars. Because the newly formed cells are especially susceptible, the growth hormones of pregnancy, puberty, and childhood increase the duration and intensity of dynamic scar build-up.[118]

Hypertrophic scars can be decreased by meticulous, gentle wound care; early, thorough wound excision by an experienced surgeon; and avoiding removal of adherent epidermal cells during dressing changes. Discontinuing topical antimicrobials that are fibroblastogenic as soon as practical, maintaining adequate nitrogen balance and zinc and vitamin levels, and avoiding wound infections also positively influence scar formation.

Effective scar management includes external vascular support, which is begun as soon as wound healing is adequate so the support does not adhere to the injured tissues[119-123] (Figure 10-82, Vascular support for (*a*) superficial burn, (*b*) superficial partial thickness burn, (*c*) split thickness donor, (*d*) deep partial thickness burn, (*e*) full thickness burn, and (*f*) full thickness donor). Unless worn only for protection, the external vascular support appliance must apply continuous pressure in a gradient manner. The pressure must be adequate to decrease capillary circulation and must be continued until the scar matures. Pressure adequate to decrease edema and compress scar tissue cannot be applied to the middle of an extremity without impairing lymphatic return. Therefore, the principle of gradient pressure, the most support at the distal limb and the least at the proximal limb, is important. The hand or foot must always be supported in some manner. During the day for instance, a laced shoe can support scar tissue and provide a satisfactory outcome. A clean shoe must then be worn at night or the vascular support fitted over the foot. Although scar tissue usually looks shiny and is lighter in color than surrounding tissue, the optimal final healed wound should appear flat, soft, mobile, durable, supple, of proper color, and have minimal thinning and wrinkling.

The healed wound, donor, and grafted areas are evaluated for application of external vascular support when open areas are smaller than 3 cm^2 on the trunk or leg and 1 cm^2 on the face or hand, and when the tissue is durable enough to tolerate the shear of garment application. A thin, moist contact layer prevents the external vascular support appliance from sticking to the epithelium at the wound edge. The elastic wraps or garment should be soaked off during bathing to prevent denuding skin. When coarse mesh gauze wrap, such as Kerlix (Kendall Healthcare Products Co.), or fine mesh gauze, such as Unna, are used as a contact layer, a nylon stocking may be used to allow donning the support without rolling the bandages. The nylon stocking is left in place under the external vascular support wrap or garment. As the wound matures, the individual stops using the contact layer, gauze, or nylon stocking and applies the support directly to the healed skin.

The most distal part of the extremity, that is the hand or foot, must be properly managed first. Even if the upper arm is developing thick, rope-like scars, the hand must tolerate support before the arm is included, and the elbow must be supported without causing damage before the upper arm is supported. When it takes several weeks to achieve tolerance of support garments for the hand or foot, the more proximal scar tissue will still respond to support and become softer and lighter in color when the full limb garment is begun.

The most universally used early external vascular support is the elastic bandage wrap, applied in a gradient figure-8 manner, from toe to groin. Since early excision and grafting has become the rule, external vascular support has rarely been provided in the acute phase of healing for the head, neck, upper extremities, and trunks. Once wound closure is complete, a wide variety of commercially available off-the-shelf support designs are available (Figure 10-83). Many patients tolerate the cotton and rubber prefabricated garments well, the scars recede and no other support is needed.[124] In addition, fabric with varying elasticity characteristics is available from most custom measured garment manufacturers, and custom fitted orthoses of multiple types are also available for external vascular support and scar compression.

Contracture and Scar Management

Head and neck. Facial skin is loosely connected to underlying structures. If this connective tissue is allowed to contract around the face or neck, permanent, grotesque distortion of the nose, eyelids, mouth, ears, and neck may result. When the wound extends into the reticular dermis and heals spontaneously, an unnecessarily bulky, hard, red scar will almost certainly occur. Similar distortion during healing is noted when the wound is on the loose anterior or lateral neck (Figure 10-84). The beneficial use of external vascular support equal to capil-

a **b**

Fig. 10-82. Vascular support after burn injury. (**a**) Shallow, superficial burn and (**b**) Superficial partial thickness burn.

c

Fig. 10-82 continued. Vascular support after burn injury. (**c**) Split thickness donor.

d

e

Fig. 10-82 continued. Vascular support after burn injury. (**d**) Deep partial-thickness burn, and (**e**) Full-thickness burn.

f

Fig. 10-82 continued. Vascular support after burn injury. (**f**) Full-thickness donor site.

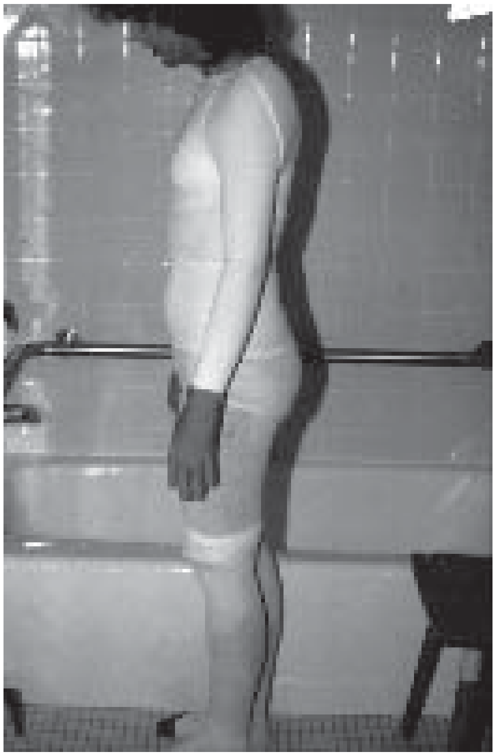


Fig. 10-83. Prefabricated external vascular support garments, Isotoner gloves, Tubigrip long sleeve shirt and long leg pants.

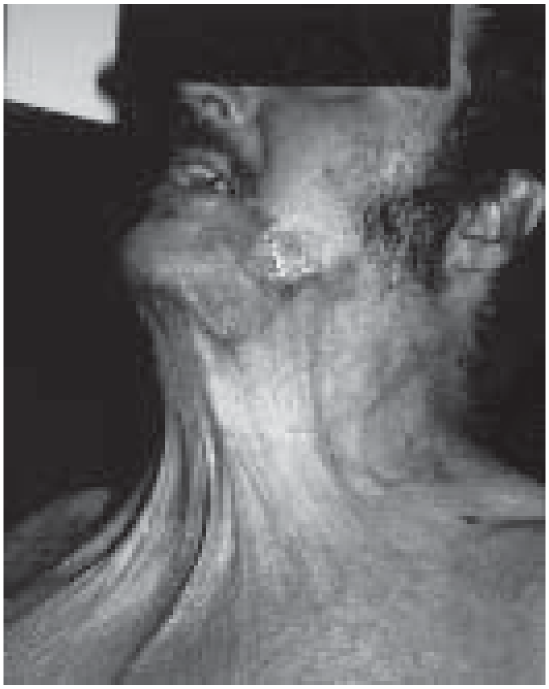


Fig. 10-84. Contractures of the neck.

lary pressure, which compresses and supports the healing burn wound, is widely utilized for the face and neck.

A variety of appliances are available to manage face and neck scars. An accurately fitting, total contact, transparent facial orthosis worn 20 hours a day during the maturation phase of wound healing is one method to prevent distortion of facial contours. A less expensive, less therapist-intensive, but less than optimal, method of modifying the scar tissue is a custom measured elastic hood that secures a custom made silicone facial insert. However, used alone, an off-the-shelf hood or chin strap or even a custom measured elastic hood are ineffective because the elastic garment cannot conform to hypertrophic scars in concave areas. Scars in concave areas, such as under the mandible, need an insert for compression (Figure 10-85). Off-the-shelf hoods can be modified to decrease perspiration by replacing the crown of the hood with X-cross elastic straps. Patients wearing external vascular supports or orthoses for the face and neck must be closely observed for complications such as sleep apnea,[125] changed bone growth,[126] and posterior migration of the teeth. Patients who have severe breathing problems may need observation for sleep apnea and oxygen saturation monitoring overnight to determine optimal safe night positioning. Sleep apnea monitors used at home are very disruptive to the family and, therefore, hiring a night attendant to check breathing at short intervals of time is more appropriate.

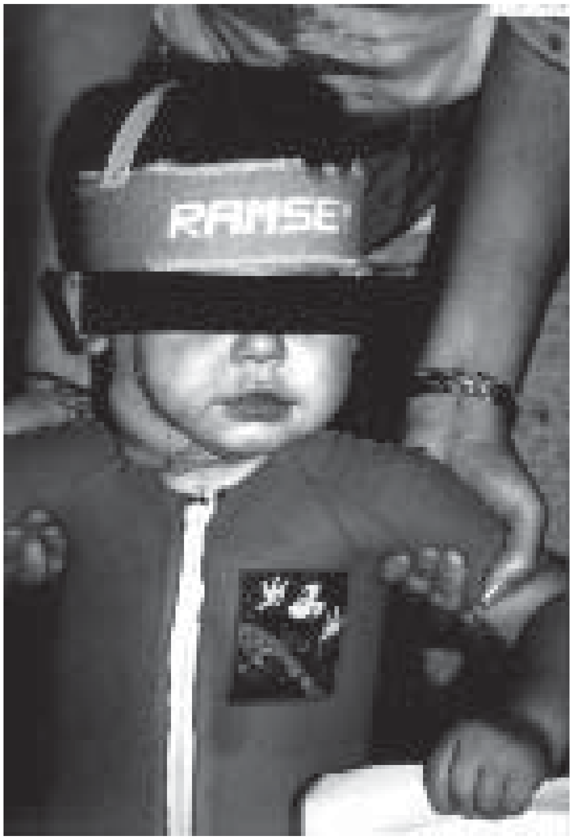


Fig. 10-85. Hood with pocket used to secure compression insert.

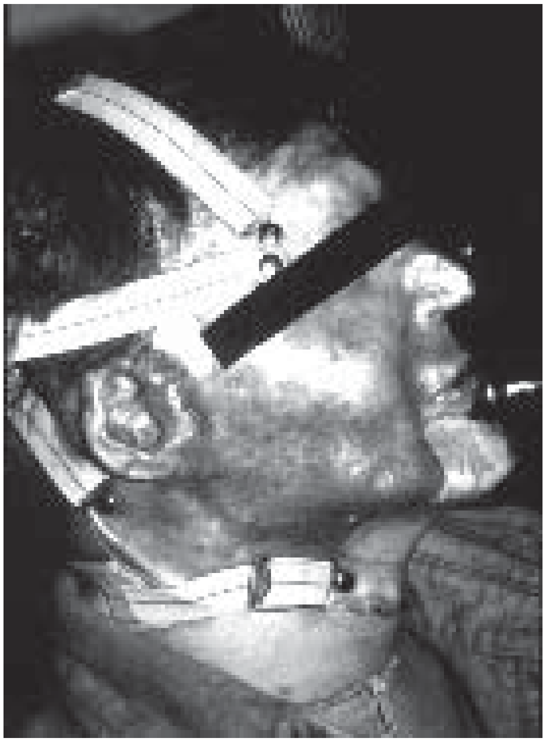


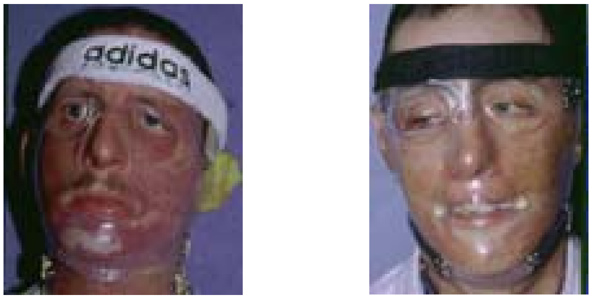
Fig. 10-86. Clear facial orthosis. Xeroform gauze is used on the chin.

A properly fitting clear plastic facial orthosis will compress the tissue against the underlying skeletal surface to preserve natural contours. The person wears a thin disposable contact layer such as Xeroform (Chesebrough Ponds, Inc.) over infected facial hair follicles (Figure 10-86). This is changed whenever the mask is removed. If heat rash develops, a thin paper towel sheet may be used and discarded each time the mask is removed. The person must learn to speak slowly and enunciate clearly to be understood while wearing the orthosis. The orthosis is removed for chewing and for oral stretching exercise.

A properly fitting neck orthosis is more difficult to achieve because there are no bony landmarks on the anterior neck, the larynx must move during

Burn Rehabilitation Outpatient Home Care Program

THE TRANSPARENT FACE MASK HOME INSTRUCTIONS



Transparent day orthosis **Transparent night orthosis**

Night orthosis is worn with mouth stretching orthosis, nose cones, and eye humidity domes, as needed for scars.

PURPOSE The splint maintains normal face contours, decreases pain and itching, prevents contractures, minimizes scars and moisturizes tissue.

WEAR Continuously, except to bathe, eat and exercise. (20 hrs/day)
NEVER LEAVE MASK OFF LONGER THAN ONE HOUR!!!!!!

APPLY ___ Directly to skin. (Kerlix patch if needed)
___ Over or ___ under elastic elastic hood.

SPLINT CARE Splint is plastic. Wipe with soapy cloth and cool water.
Rinse well.
Dry thoroughly before re-applying.
Clean splint hourly, decreasing to daily.

SKIN CARE Observe face for swelling or poor circulation. Observe skin under splint for reddened areas and call therapist if rash or breakdown is observed.
Do exercises thoroughly before applying splint to prevent rubbing.
Wipe splint and skin dry as needed. Re-apply splint quickly.
Normal skin will gradually decrease perspiring. Burned skin will not sweat.
Do not perforate splint in any red areas!!
Wear gauze or towel to keep skin dry, if needed.

ALWAYS BRING ALL SPLINTS AND MOUTH INSERTS TO CLINIC!

Problems with open areas or infections must be referred to the doctor, clinic, and therapist.

If patient will be in sunlight, wear sunscreen under splint when removing splint for exercise.

Have teeth checked by a dentist or orthodontist prior to beginning to wear this splint. Dentist or orthodontist should check for tooth alignment, decay, and hygiene at least every 3 to 4 months and up until 6 months after wearing the orthosis.

SPLINTS ARE FLAMMABLE. DO NOT EXPOSE TO SOLVENTS, OPEN FLAME OR CIGARETTES.

Fig. 10-87. Burn rehabilitation outpatient home care program. Reprinted with permission of Regions Hospital (formerly, St. Paul-Ramsey Medical Center), 640 Jackson Street, St. Paul, MN 55101-2595.

speech or swallowing, and the sternocleidomastoid muscles change as they contract and relax for neck movement. However, with practice, a neck orthosis may be fitted that reduces edema and supports the tissue between the sternal notch and the chin at the maximum possible length. Neck hyperextension alone will not prevent neck contractures. Because the shortest distance between two points is a straight line, the neck skin migrates anteriorly and forms a neck contracture with bands around the neck. The night neck splint incorporates some hyperextension. The head is in the neutral position for the day neck orthosis so the person can read and walk safely without having to bend forward causing strain at the hips or back. The day position resembles "hanging" from a rope under the chin.

Written wearing instructions are given to each wearer (Figure 10-87). Some individuals ask for wallet identification cards indicating the facial orthosis is a medical necessity. If questioned by authorities, this card can identify a resource to call for verification that the person needs the splint.

Although orthotic use is very helpful to achieve an optimal outcome for the maturing face or neck burn, the orthotic device alone will not create a mobile, supple scar. Wearing an orthotic device that causes decubiti increases the inflammatory process and worsens final outcome. The wearing of an orthosis never replaces AROM to improve the quality and length of the healing epithelium and underlying connective tissue. Wearing a facial orthosis can exacerbate oral contractures. Syringe cases assist the patient to stretch the horizontal oral opening to the same distance every day (Figure 10-88). Proper use of nongreasy sun screen and the use of adequate medication to decrease pruritus assists the individual to avoid damaging newly healed tissue. Exercise (see Figures 10-69, 10-88, and 10-89) reduces facial and neck edema, meticulous hygiene decreases the inflammatory process, and gentle massage prevents selective tethering of the skin to the underlying wound or muscle. When the skin is tethered, it wrinkles and makes the mature scar obvious to an observer.

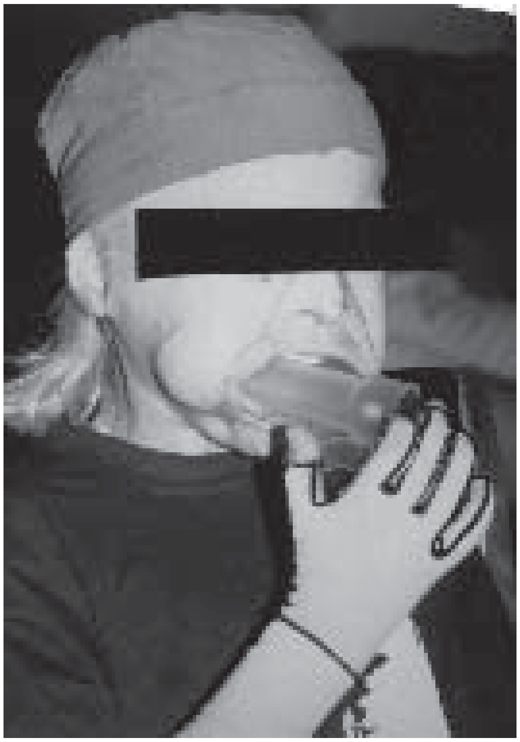


Fig. 10-88. Mouth stretch using syringe cases.

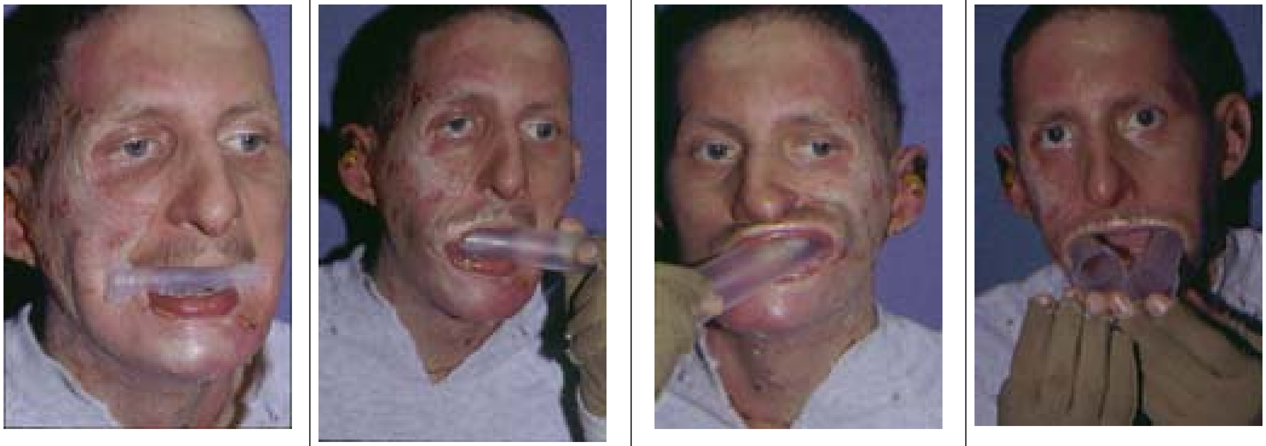
After the tissue becomes light in color and the scar is mature, the orthotic support is discontinued. Because the capillaries have not developed normal tone, there is an initial hyperemic response. The likelihood of scar build up after the face or neck orthosis is discontinued can be decreased by initially having worn a properly fitting orthotic device, having had frequent revisions made as the wound changed, and having continued use of the orthosis until the scar was mature, and then by continuing frequent follow up visits to review wound changes.

Fitting a casualty with a transparent total contact orthosis is a four-step process. The first step is taking a negative impression of the face or neck. The whole process of taking this impression requires approximately 15 to 30 minutes for an experienced therapist or orthotist. Scalp hair is contained in a surgical cap and this is taped in place. Hairy surfaces such as eyebrows are coated with petrolatum to facilitate moulage removal. Alginate dental impression material, prepared in cool water, is spatulated in a flexible bowl until the mixture is bubble free. It is poured over the face, leaving an oral or nasal airway. Some centers use Duplicast[127] instead of alginate for the contact layer of the moulage. In this case, the skin must be well lubricated to allow removal of the moulage. A gauze strip may be imbedded as the alginate or elastomer sets, before the entire moulage is reinforced with four layers of fast setting plaster bandage strips. The reinforced moulage sets in two minutes, the soldier sits forward, inhales, and gently puffs air into the closed moulage to break the vacuum. With a gentle tug on the adjacent skin, the moulage falls away from the face. Care must be used to return the back of the model to its original dimensions, or the positive plaster cast will be wide at the back of the jaw and ears, causing unnecessary carving for proper fit of the mask. Some centers use silicone elastomer to take facial impressions. It is important to practice with the catalyst and elastomer to assure the material has completely set-up. Removal of liquid elastomer is complicated, can destroy fragile skin, and is painful.

The neck moulage is taken by massaging plaster strips around the scarred areas of the neck, leaving a back opening for removing the plaster from the neck. The neck and any hairy areas to be included

DO EXERCISES AT LEAST FOUR TIMES EACH DAY

(1) Remove splint. (2) Exercise. (3) Wash skin and splint. Dry well. (4) Reapply splint after exercise. (5) Do not leave splint off longer than 10 minutes. (6) Repeat exercises for both sides of face. (7) Hold each stretch at least for 2 minutes. (8) When skin will tolerate more stretch, increase case size.



To keep facial tissue loose and mobile, *slowly stretch* the cheek by placing a syringe case between the teeth and into the pouch of one cheek. The corner of the lips on the opposite side of the mouth is also stretched.

Stretch both corners of the lips backward. Pull them into a large "EEE" by placing the syringe case between the teeth and pulling backward at the corners of the mouth. This is the second exercise to do with the cone-shaped stretching device or syringe case.

The third exercise stretches the corner of the mouth. Slide the cone between the cheek and the teeth. This stretches one side of the lips and cheek pouch. Hold the stretch on each side for a count of 60.

The fourth stretch uses two cones, one in each side of the mouth. Slide one syringe case between teeth and corner of lips on the most difficult side. Then slide the second device in, crossing the syringe cases at first. Both corners of the lips and both cheek pouches stretch. Hold for a count of 100 or longer.

Fig 10-89. Face exercise program for home care of burn rehabilitation outpatient.

are well lubricated with petrolatum, or an equivalent, to facilitate removal of the negative mold without removing hair. Two or three layers of plaster strips are placed in progressive layers from the lower lip and ear area to 1 in. below the sternal notch. The first strip is massaged into place around the ears and chin, then the second is attached, and then the third is placed over the lower neck and clavicle area and folded into a "V" shape to conform to the anterior neck. This negative mold is removed and used the same way as the alginate and plaster facial mold.

The second step in fitting a casualty with the proper orthosis is forming a positive plaster cast of the casualty's face or neck. The positive plaster cast takes approximately 20 minutes to finish, depending on the setting time of the plaster. The following materials are needed:

- 12-minute impression plaster,
- plaster spatula,
- mixing whisk,
- Flexible, 4-in. angled reinforcement bar or bolt,
- petroleum jelly,
- a plaster stand,
- a clay modeling tool or tiny spoon,
- file,
- sandpaper,
- fiberglass sanding strips,

- cloth strips; and
- a vise.

Openings for the nose or mouth are closed with Jeltrate (LD Caulk Division) and supported with plaster strips. The bowl shape is formed with plaster strips over a paper towel to close the top and the bottom of the form. Bubble-free impression plaster is mixed in equal parts of water to plaster and is poured immediately onto the lubricated negative impression. This positive cast must be made immediately because alginate shrinks rapidly once mixed with water and exposed to air. When the anterior or deep portion of the mold has firmly set, the rest is filled with plaster and an angled bar or bolt is incorporated into this part. The mold sets in 20 minutes, whereupon the bar is clamped in a vise and the alginate and plaster are gently separated. Special care is needed to remove the alginate over the nose area because the poorly reinforced protruding nose breaks off easily. A paper clip can be dropped into the plaster to reinforce the nose, but this may complicate carving later. The neck is prepared in the same manner, except the plaster is more likely to adhere to the positive cast without the alginate interface, and additional care must be used during separation.

The bar or bolt is firmly clamped in a vise and the surface of the plaster model is painstakingly smoothed with a modeling tool, a small spoon, and Durite cloth. The hypertrophic scar details are marked with marking pen and smoothed off in the plaster mold so that the plastic mask will provide adequate total contact pressure to the maturing face or neck wound. Finally, the mold may be buffed with a cloth strip to provide a polished surface over which the plastic mask will be shaped. This total process takes about 30 minutes.

The third step in fitting the orthosis is fabricating the transparent plastic orthosis over the finished plaster cast. This may be done with a vacuum former or by hand, gently pressing the plastic over the plaster. This process takes 15 minutes, more or less, depending on the therapist's past experience. When tools fail or if the plastic bubbles, completion time is extended. Materials needed include:

- a 25.40 x 30.48 cm sheet of copolyester,
- a sheet of clear plastic,
- a toaster oven (204°C/400°F dry heat),
- a heat gun with spot-heater attachment,
- metal files,
- 3-0 steel wool,
- utility knife,
- felt cutters,
- scissors,
- Dremel motor tool with cutting blades and sanding drums,
- a round punch or drill press,
- four elastic Orthobands,
- four rivets,
- hammer,
- anvil,
- vise, and
- two pairs of double gloves.

The sheet of copolyester is heated in an (204°C/400°F) oven for 1 to 5 minutes and gently draped over the mold while two people carefully depress the plastic into the contours. This must be done quickly since the plastic cools in less than 1minute. Orthotics laboratories do this with a vacuum former. The excess plastic material is then removed and openings are cut for the eyes, nose, and mouth. The straps are attached in an "X" pattern from the temple to behind the opposite ear with the top of the "X" anterior to the crown of the head. This total process can take from 30 minutes to 3 hours.

The fourth, and by far the most difficult step in this fitting process, is revising the mask to be certain of accurate total contact pressure on the healing tissue. This is time consuming. It may take several hours. Sometimes the center of the face is all that can be fitted in one visit. Revisions of small areas must be done at each visit. Some less active scar areas can wait until later visits. As more dense scars soften and thin, these areas are revised for improved pressure.

Once the orthosis is strapped in place, the evaluation for revisions and fit begins. Areas of the hypertrophic scar are observed through the plastic. If they do not blanch, the areas are marked, transferred to the plaster model, and the plaster is carved down to develop gently rolling final contours. The plastic orthosis is changed until all scar tissue is flattened against the underlying bony contour. Scarred areas that need additional pressure can be revised by cutting away the plaster and spot heating the plastic with a heat gun. The transparency of the plastic mask permits continuous assessment and precise remolding. If the scars are very irregular and thick, the plaster positive must be gradually shaved off so the plastic mask will provide constant, total contact pressure. Excessive pressure applied before the scar has begun to soften can cause ischemic necrosis of the tissue. Too much undistributed pressure in the areas overlying the gums can irritate gum tissue. The pressure should be applied in a way to "capture" the scar and distribute pressure evenly over the total scar, not just in the center of the scar. The plastic will tolerate at least four to six reheatings for revisions before it bubbles or becomes too brittle or too flexible to apply adequate pressure. Initially, the revisions for total face contact are done weekly; as the scar tissue matures, longer intervals are possible. A new plaster mold is needed only if head growth takes place or the plaster positive has been carved beyond repair.

The mask must be worn at least 20 hours a day to attain the desired result. A plastic orthosis that does not conform to the contours of the underlying skeletal structure is not likely to control the hypertrophic scar activity even if it is worn 20 hours a day. Although patients sometimes leave the orthotic appliance in a drawer and expect scar relief by wizardry, wearing the splint for a year or more is the only successful way to modify the scar.

If possible, when the entire face is scarred, it is preferable to control the forehead scars with a wide elastic head band; and the nose, cheeks, and chin with the transparent facial orthosis. This technique permits more freedom for motion of the jaw and facial muscles and less shearing force on the healed skin when the patient talks.

The casualty may require an orthodontic retainer to prevent posterior displacement of the teeth. If microstomia is a problem, the patient must also wear a mouth-stretching device. At least 4 half-hour exercise periods must be set apart each day for the patient for exercise. The mask must also be removed for 3 half-hour meals daily and for a 30-minute shower. Patients are encouraged to exercise and eat in 3 one-hour periods at the usual times, and to bathe and exercise before bedtime. The mask is never off for more than 1 hour at a time. Patients, family, and nurses need a written wearing schedule and instruction card (see Figure 10-87).

A special orthosis may be fitted to keep the nares patent. Custom fitted inserts may be made of soft silicone or hard acrylic. A separate nose orthosis may be fitted under the clear facial orthosis to maintain patent nostril openings.

If the ear meatus is scarring closed, it can be maintained with a silicone elastomer insert or a hard plastic or acrylic insert. An "Oyster shell"[128] orthoses can be fitted to preserve the upright pinna of the ear and to reduce hypertrophic scars over the outer ear. These are very complex and difficult orthotics to fit and wear.

There are several prosthetic companies that fit prosthetic noses and ears as well as other parts. The Life-Like Laboratory, 2718 Hollandale Lane, Suite. 400, in Dallas, Texas 75234, will provide these by mail order if an accurate model of the patient's head, color photographs, and a prescription is supplied.

Hypertrophic Scar, Edema, and Contracture Management of the Thorax and Limbs

As soon as the wound or donor tissue have no open areas larger than 3 cm^2, external vascular support management of edema and scar compression is initiated (see Figure 10-83). Although early continuous vascular support for hypertrophic scar treatment is well accepted, debate continues regarding the optimal pressure for these supports.[129] The majority of literature indicates that 25 mm Hg is necessary for scar treatment in order to exceed the level of capillary pressure.[130] The lowest was reported at 4 mm Hg to 5 mm Hg. Some clinicians stress that correct measuring of pressure in mm Hg against varying density of bone, muscle, and fat and differing body surface contours is impossible. Pressure measurement is probably not as important as having a fit that controls the scars and does not cause decubiti or nerve damage. It is suggested that children need less than 10 mm Hg to 20 mm Hg. A patient should be given the least pressure in the support that will decrease edema and modify the scar. Friction and shearing, especially at the elbows, axillae, and knees causes increased fibrotic reaction; chronic open areas develop and final wound resolution is slowed. Adequate support is postulated to enhance progressive devascularization of hypertrophic scar tissue and to speed wound maturity.[118-120]

The initial purpose of the external vascular support is to protect fragile, newly healed skin from blistering; improve venous and lymphatic return; decrease extremity pain; decrease itching; prevent sunburn or frostbite; moisturize epithelium; modify overly bulky, thick, hard, scars; and elongate maturing contracture bands. Complete baseline descriptions of the healed wound, any open areas, and areas of early scar symptoms or contractures are documented. Patients usually have signed a release for photographs, which become a part of the medical record. These photographs are taken at regular intervals to reveal scar changes. The least abrasive external vascular support is initiated to increase the durability of the epithelium. Gradually, tighter supports are introduced until maximum scar control is achieved without circulatory or nervous system complications.

External vascular support is initiated in the posthealing phase when staples are out, grafts are stable, and most open areas are dry. Gradient elastic wrapping progressively decreases the rate of applied pressure from the bandage and is changed according to a variable magnitude.[131] Elastic bandaging that is the tightest at the distal end and loosest at the proximal end of the limb simulates the pressure of water against a limb when vertical in a swimming pool, which pressure will improve venous and lymphatic return. This kind of bandaging can prevent hemorrhage of granulation tissue or hematomas under recently adherent STSG, and it will relieve blistering on dependent limbs (see Figure 10-27).

Generally, burned legs have been supported with figure-8 elastic bandage wraps from the acute injury through discharge from the hospital to facilitate ambulation, prevent pain, and lower extremity edema or hemorrhage.[106] These elastic wraps are applied while the soldier is recumbent, to avoid edema and poor venous return when standing. In cases of severe edema or poor quality elastic bandages, double bandages are used. Studies have shown early ambulation on the third to sixth day after grafting is safe, using an Unna support, which is a fine mesh, nonbias gauze medicated gelocast dressing impregnated with zinc oxide[132,133] (Figure 10-90). Wrapped in a spiral or figure-8 pattern around a lower extremity graft, a nearly healed burn or a donor area with small open areas, the Unna ([ConvaTec] ER Squibb & Sons) dressing will dry the wound, accelerate healing, and provide durable support and protection. An Unna dressing can be worn for 3 to 7 days in between changes. When fingers or hands are edematous and draining, they may respond to early edema management with Unna dressings along with elevation and exercise. When present, open tendons should be protected from the drying of an Unna dressing by a contact layer of Duoderm ([ConvaTec] ER Squibb & Sons) or other moisturizing dressing and the dressing must be changed daily. When the extremity is healed, it is usually durable enough for tubular external vascular support to begin on a foot and leg or Isotoner gloves on a hand. The prefabricated garments are made of a variety of materials from unidirectional, stretchable, rubber, elasticized cotton, to elasticized nylon, nylon/spandex, spandex, and rayon.[122]

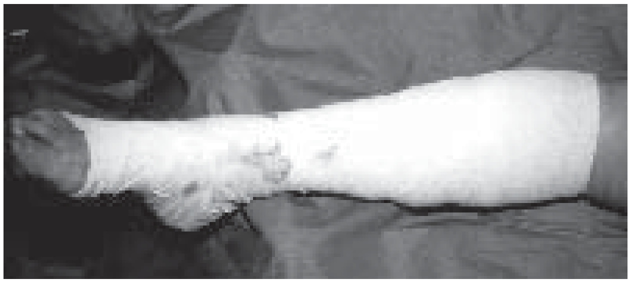


Fig. 10-90. The lower extremity, wrapped in a Unna dressing, is ready for the elastic wrap.

The casualty is given written directions for care of the wound and the external vascular supports (Exhibit 10-4). Elastic bandages are hand washed, rinsed well, and hung or laid over a net to dry. Some supports can be machine washed and dried. Others are hand washed in a mild soap (such as shampoo but not detergent), rinsed thoroughly, and air dried. The second support is worn while the first one is being cleaned. It is helpful to date or number the supports to keep track of their age.

Skin covered with support garments may develop offensive odors. Daily bathing, washing open areas and body wrinkles thoroughly, drying skin meticulously, and applying a lotion every day improves wound hygiene. Lotion should be rubbed in well prior to donning the support garment. Heavy petrolatum or oil-based lotions should be avoided because they liquefy natural sebum and it is then washed away. Wearing deodorant is appropriate if it does not cause contact dermatitis. If blisters form or open areas increase in size, discontinue the support garment, resume elastic wrapping of the extremity and explore the causes of blistering. If excoriation is from scratching, increase or change the antihistamine medications. Continue ambulation and exercise. If movement is causing open areas, exercise may be done in bed in an elevated position before the elastic wraps are applied until tissue becomes more durable. Drain the blister from the edge if the area is spreading from the pressure of elastic wrap. Drained seromas or hematomas need a pressure dressing to prevent them from reforming. Apply a small dressing over the blister, under the garment. It may be necessary to remove elastic wraps when the casualty is lying down to help open areas heal. Once they heal, resume 23-hour-a-day use of the external vascular support garment. It is normal for casualties to develop blisters for several months because absent dermis does not regenerate and fragile skin very slowly develops rete pegs, which increase durability. Attempt to continue the garments as tolerated. The garments are applied directly to the skin as soon as it becomes durable enough to tolerate them. Very fragile skin may be painted with Merthiolate or tincture of ben-

EXHIBIT 10-4

OUTPATIENT HOME CARE PROGRAM: TUBIGRIP EXTERNAL VASCULAR SUPPORTS

PURPOSE: Compression sleeves, stockings, and garments support newly formed blood vessels; prevent swelling; decrease itching; decrease the thickness of scars; help scars elongate; help moisturize skin; and protect fragile, newly healed skin from blistering. Tubigrip is a rubber and cotton material that comes in either presized tubular rolls, premade garments, or shaped support bandages.

WEAR: Continuously, all the time except during a bath or when applying lotion. Never leave off more than one hour.

APPLY:
____ Directly to skin
____ Over Kerlix wrap
____ Over nylon stocking interface
____ Over/under custom measured elastic garment.
____ Small amount of ________ to open area, gauze patch, then nylon and then "tubi"

WASHING: Wash tubis daily, by hand, in warm water and mild cleanser such as shampoo. RINSE WELL. Air dry or use dryer on air fluff. Rubber deteriorates when exposed to oils or extremes of heat.

SKIN CARE: Keep skin very clean and dry and moisturize with lotion every day. If blisters form or open areas increase in size, discontinue using the Tubigrip™ cylinder. Resume elastic wrapping of your extremity until checked by your doctor.

PRECAUTIONS: Shearing of the fragile skin as the Tubigrip™ is donned should be avoided. A coffee can may be helpful to stretch the tubi as it is donned. Do not wear the tubi if it is causing sores. Padding usually makes the tubi tighter, so check with the therapist before adding padding. If scars are getting thick and hard, check with therapist or doctor. Elastic wrap over the tubi until you are seen in clinic. Tubigrip cylinders must often be worn in double layers to adequately compress the tissue. If your extremity is swelling, report it to your doctor at your clinic appointment.

If you need more Tubigrip, inform the clinic nurse. Be sure the therapist has checked the fit before you order more Tubigrip, especially if your scars are hard and dark red. Be sure you get the correct size. The tubular rolls are available in sizes A through G and J through L, the Tubigrip garments come in sizes 1 through 5. Shaped support bandages come in 6 sizes.

Make a list and pick up enough supplies to last until your next clinic visit. If you are having problems with supplies ask your Qualified Rehabilitation Counselor, your Medical Assistance Financial Worker or the Burn Clinic Social Worker for assistance.

For problems with scars, contact ____.

For problems with blisters or infections, discontinue using Tubigrip, elevate extremity, and contact your doctor.

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zoin to dry and toughen the area.

The supports are removed while bathing and occasionally during a rigorous exercise program. If they are left off for more than 1 hour, the area should be rewrapped with an elastic bandage. The most distal part of the limb must always be supported, so a glove, for example, cannot be removed for exercise or to view the wound unless the sleeve and axillary supports are also removed. Support for the thigh is initially accompanied by support toe to groin. When using Biker's compression shorts (Figure 10-91) for thigh donors or grafts, it is necessary to observe the ankles for edema because this support may have a tourniquet effect on the distal limb.

The external vascular support garments are usually worn for 12 to 24 months with an average time of 18 months. If the scar process worsens, inserts or overlays are added. Eventually all scars begin to

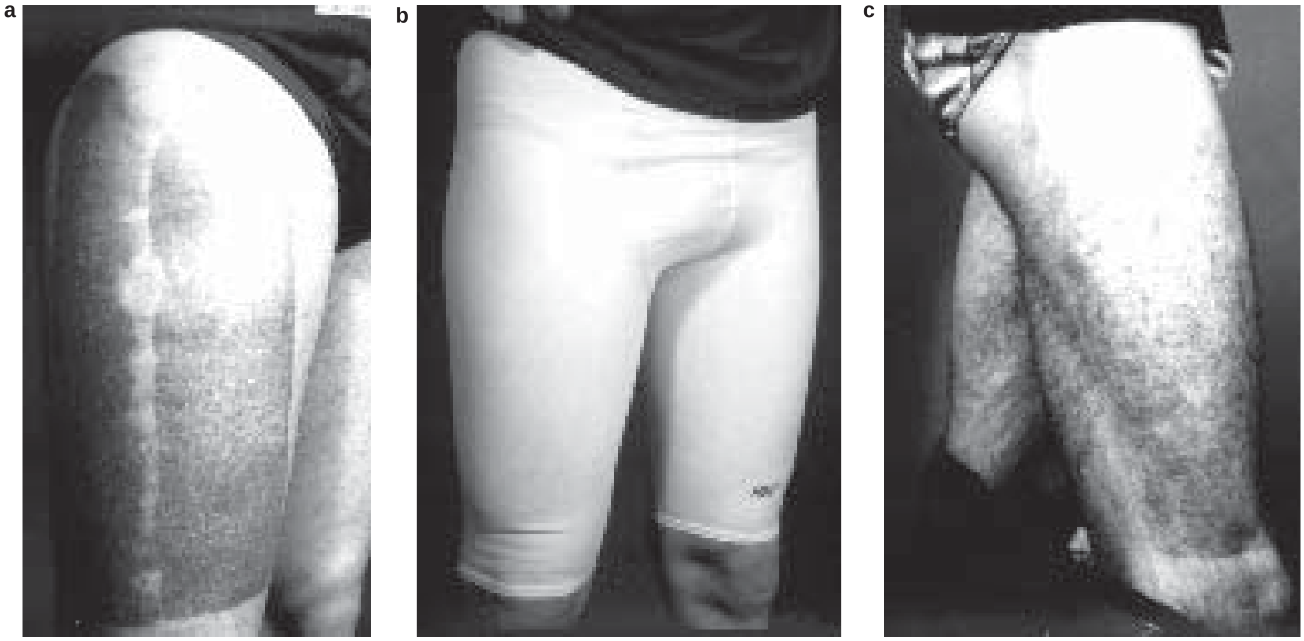


Fig. 10-91. Good donor outcome with biker's compression shorts. (**a**) Immature donor. (**b**) Biker's pants. (**c**) Mature donor.

regress (see Figure 10-82). When the healing wound becomes soft, of proper color, and flexible, the garments are discontinued for 3 to 7 days. If no changes in scars are noted, the wound is mature and the external vascular supports are discontinued. There is a hyperemic rebound response during the first 7 to 21 days after the support is discontinued. Initially it may be necessary for the soldier to wear the garments when ambulating and discontinue them when in bed. When stinging and itching are noted during ambulation or standing, the support stockings are continued at those times. If edema and the scar process re-forms, the supports are resumed for another 3-month period.

For external vascular support of the hand (see Figure 10-46), Isotoner gloves are an economical off-the-shelf type of support glove which is manufactured in three sizes for hands that are not extraordinarily large or small. Elset (Seton/Tubiton House) is a lighter type of low tension elastic bandage that allows freer finger movement. It can be wrapped in the figure-8 "boxer" wrap when the soldier has adequate strength to make a fist against the elastic wrap tension.[134] The fingers can be individually wrapped in a variety of 1 in. spiral wraps such as Sof-Kling (Johnson & Johnson) or Unna. Coban (3M Healthcare) is a light crepe self-adherent elastic wrap that supports fingers well but can become too tight (Figure 10-92). It also has a light adhesive that sticks to whatever it comes in contact with. Several compression companies provide elastic digi-sleeves, also called edema-sleeves. A thicker protective foam called LMB (North Coast Medical) finger pressure wraps can be applied with Velcro. A slip-on sleeve can be made by sewing a small section of Ace, cotton elastic, or spandex fabric. Tubiton is a finger stockinet that can be doubled over the finger to take up the shear when applying a glove (see Figure 10-42). If the hand itself is not ready for a prefabricated glove, it can be wrapped with Unna, PEG self-adhesive bandage, Coban or other elastic wrap. The fingers of the glove can be cut off, and it may be used as a gauntlet. The glove can be modified by splitting the dorsal aspect or side and sewing in Velcro for ease of application. Several companies provide prefabricated, variable-sized gloves, sleeves, vests, and pants. The amount of tension can be easily altered with a serger sewing machine.

Prefabricated garments, such as the Tubigrip (Tubiton House) line, provide external vascular support to the trunk and limbs (see Figure 10-83).[124] Measurements for a prefabricated tubular compression garment for an extremity or the trunk are taken from the widest circumference of the body part, and the appropriate width tube is selected.[135] Tubigrip provides wide tape measures that give you a choice of low, medium, and high tension selections. Experience indicates low tension improves durability of fragile epithelium, and high tension is appropriate after distal extremities are tolerating high tension

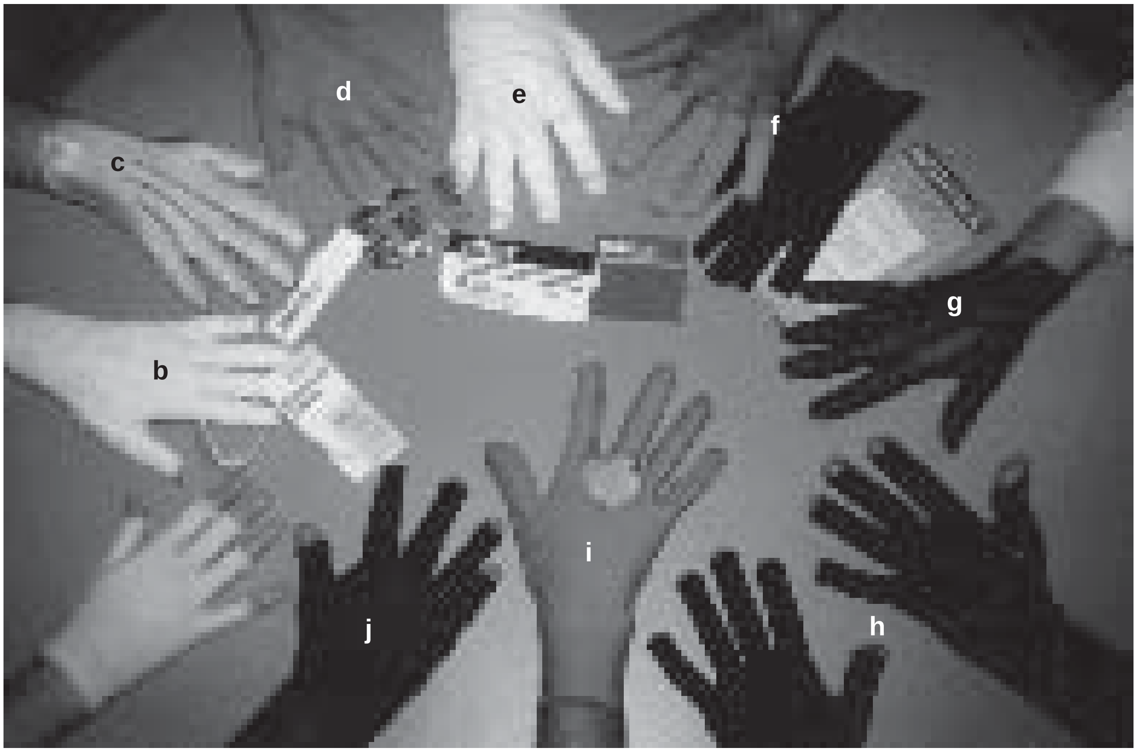


Fig. 10-92. Hand external vascular supports: (**a**) blue soft fabric custom measured Barton Carey glove; (**b**) spiral Unna finger and hand wrap; (**c**) Jobst Interim glove; (**d**) Coban spiral finger wrap with dorsum of hand included; (**e**) Elset "Boxer" wrap over dorsum of hand; (**f**) Tubiton Oedema sleeves on black Isotoner glove; (**g**) Gray Isotonor driving glove; (**h**) Palm of leather work glove; (**i**) Barton Carey custom glove with soft fabric over MCP; (**j**) Insert stabilizer glove with seamless thumb design.

support. Straight tubes are often doubled over with the shorter layer being next to the skin and a slightly longer layer on the top to avoid a tourniquet effect when they both end at the same place. If the extremity is a variable circumference, as in a heavy person or with someone with well defined musculature, a tapered or shaped support bandage can be used. The regular tube can also be put on in a single layer in the area of wider girth and doubled over in the thinner area. The tubes come in rolls and are cut to the desired length. A lightweight cardboard strip inserted into a wide fold at the proximal end of a Tubigrip tube decreases the risk of proximal rolling, which may cause blisters or the tourniquet effect. When the patient has large open areas and the dressings fall off the trunk or legs, a nylon stocking can hold the dressing in place for donning the lightweight tubular bandage. Tubular garments can be placed on a metal applicator or coffee can (Figure 10-93). The can is brought over the extremity. At the proximal end, the elastic tubular support is gently taken off the applicator as the applicator is moved distally, leaving the elastic support on the extremity. The garment can be doubled back up the arm proximally if additional pressure is needed. If the skin is durable and able to tolerate the shearing of applying the garment without an applicator, the garment can be applied like regular clothing. An alternate method is to leave the distal portion of the garment like normal and turn the rest of it inside out. Then pull the garment up an inch at a time, pinching the sides and bringing it right side out as it is applied proximally. A vest would be put on as a jacket.

Weight gain and loss should be stabilized before measuring for custom garments. This may take as long as 6 to 8 weeks. Custom garments are usually measured circumferentially every 1 to 1½ in. for the extremities (Figure 10-94) and at the waist, hips and chest for the trunk; the measurements are noted on the company's ordering form. Joints are marked on the circumferences record and measured on the pictorial form. Usually, hands and feet are drawn. Each company has its own forms, tapes, and style of measuring. Tape measure designs include longitudinal paper tape with many cross-sections taped on the extremity or a light adhesive measuring device placed

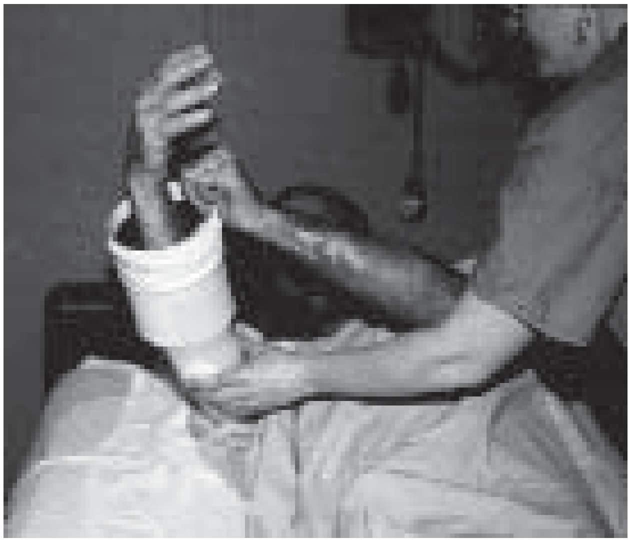


Fig. 10-93. Donning prefabricated Tubigrip sleeve using an applicator.

directly on the body. Some tapes are circular and others may be weighted. Measurement information can be quickly and accurately communicated by facsimile transmission. If a photocopy copy of the hand is used, it is sent by ground mail and facsimile, because on the original copy of the hand, what you see is exactly what the hand measures; facsimile copies distort this sizing slightly.

Accurate measurements are taken by an OT or PT, a seamstress, or a company representative. In-depth knowledge of the patient's injury and course of recovery helps plan the proper design and individual options.[136] Climate, employment conditions, physical limitations, and psychological status also influence the garment type.

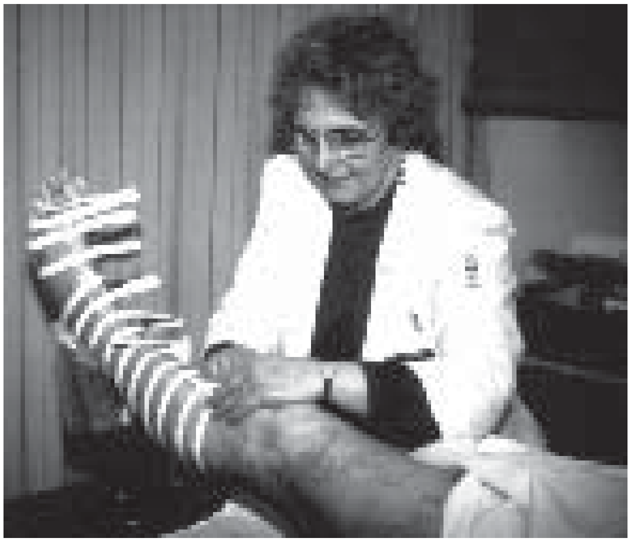


Fig. 10-94. A casualty is being measured for a garment using the Jobst custom stocking measurement system.

Patients should be measured as early in the morning as possible when extremities are the least edematous. Measurements are taken in direct contact with the patient's skin, not over clothing or dressings. The tape measure should be placed firmly but not tightly. When there are two adjacent garments, that is, a glove and an arm sleeve, overlap measurements so the garments themselves overlap and do not gap or pinch. When doing a tracing, one should use a thin marking pen. For some digit amputations that are hard to measure, a negative impression can be taken, and a positive plaster model can be made and sent off to the company with the other measurements. In these models, a paper clip or other stabilizing piece of wire should be included in the fingers to improve durability if plaster, not dental stone is used for the model. Most companies employ a bioengineer to calculate the correct formula to apply accurate compression. The garments are then produced and mailed back. The soldier tries the garment on at the therapy department (Figure 10-95).

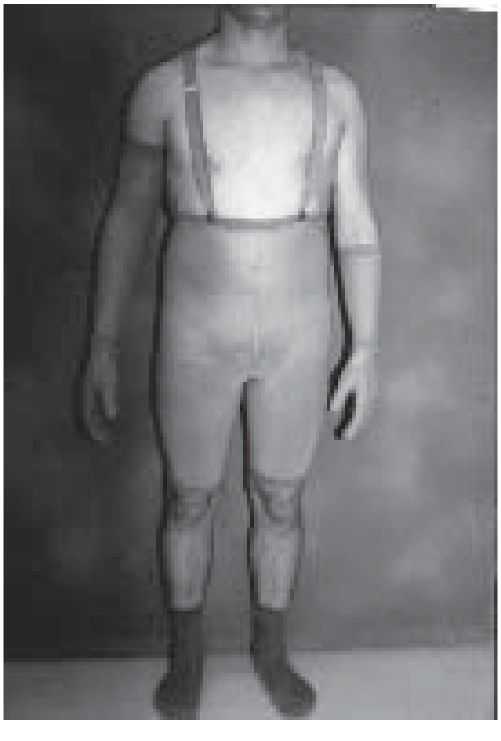


Fig. 10-95. Custom measured external vascular support garments (gloves, sleeves, and brief with suspenders).

Variables that influence garment design are the burn site, the age of the patient, the burn depth, the presence of STSGs, and the length of time to wound healing. Multiple garment designs are offered by manufacturers. Of primary importance is predicting whether a wound will scar and the location and size of the scar area. The garment should completely cover the potential scar area with a 2- to 3-in. overlap at either end so that when the patient moves, the garment will still be in contact with the area being treated. Edema and deep vein thrombosis are always a concern, therefore, the garment design must include tissue distal to the burn. Fingertips and feet can be either open or closed, depending on the patient's needs. The toe covering can be of extra soft material, enclosed as a unit or separated individually. Netting can be used to surround unburned areas for ventilation. Liners can be placed around joints with bony prominences.

The garments need firm attachment points so they do not roll, slide down, or ride up. The points could be at the waist, forearm, shoulders, or hips. Ending the garment on the muscle belly should be avoided as the edge may constrict when the muscle is flexed. Ending the garment directly on a joint can decrease circulation as well.

For the hand, the initial custom measured full glove with closed or open finger tips, slant interdigital seams, and a thumb design that allows radial and palmar abduction without losing fit over the dorsum of the hand is appropriate. Often the initial glove is made of soft fabric; the density of the fabric is increased to heavy duty when the skin can tolerate this. A leather palm work glove is also available from custom glove providers. Later, a gauntlet or mitten style design may be used if fingers are not involved. A variety of interdigital and thumb web space adaptations are available from custom garment companies. Soft fabric may be used around especially fragile areas such as the middle MCP joint (see Figure 10-92).

For the thorax and limbs, a few basic designs can be varied in myriad ways. The thorax garment can be a basic sleeveless vest; or a full suit with long arms and thighs with a crotch that is opened, closed, or has a Velcro, zipper, or snap flap. If the vest rides up, not covering the lower trunk and a body brief is not desired, the vest itself can be made several inches longer or a heavy duty snap or Velcro closing crotch strap can be added. Vests can have anterior or posterior closures of Velcro or a zipper. Velcro allows for some adjustment in the tightness. In a female, breast cups are measured and a front closure is used. A bra can sometimes be sewn into the garment instead of the compression fabric. Soft tank-top type fabric may be comfortable for unburned breasts. An unburned arm can have a short sleeve. Sleeveless vests tend to have little pressure on the chest or upper back secondary to the open arm holes. A vest with only one arm will pull the neck opening away from the open side, take away the chest and back pressure, and exaggerate poor posture. This is contraindicated. Various lengths of arm sleeves are feasible. However, if the shoulder requires compression, a vest style is more effective; and attachments keeping the waist from riding up to release pressure over the top of the shoulder is important. If the axilla is irritated from a vest that rides up, a body brief or a body suit is recommended to prevent this pinching. The axillary area may also be made larger if no compression is needed there. Pants can be waist high but if the pants fall down several options are available. Clip-on wide suspenders can be used, or a wide elastic band may keep the waist from rolling down. If a vest is also required, overlapping Velcro tabs or heavy duty snaps can be used. For bilateral thigh-high stockings, a garter belt can be used. A garter belt will not work on a unilateral thigh-high stocking. If the skin is durable enough, some skin adhesive or foam tape can be used. Knee-length stockings and anklets usually stay up well, especially if the skin is well moisturized before donning the stocking. Zippers can be placed in any of the extremities to assist initial donning and doffing when the skin is still fragile. Zippers decrease the uniform compression of the garment and at times need to be padded; it is best to avoid them if possible. Other options to add to compression garments are inserts, gussets, pads, and darts.

The initial fitting of a garment should always be done in the clinic rather than mailing the garment to the patient. This assures an accurate measurement and fit. To be therapeutic, the correct level of compression should slightly blanch the hypertrophic scar areas. There should not be any restrictions in motion, compromise of circulation, or skin integrity. Color, motion, and sensation should be checked before the patient leaves the clinic. The fingers and toes should be observed for swelling, coolness, and duskiness, and the patient should be asked about numbness or tingling. The patient should be instructed to discontinue the garment, reapply elastic wraps, and call the therapist if problems occur at home.

Applying the new garment for the first time is challenging. It should fit tightly like a wet suit, and it should take several minutes to don it. A child may require a back closure to keep the garment on. Children will naturally require assistance for donning and fastening the closures. If the garment is a trunk tube it can be stepped into, pulled up to the axillae, and sleeves pulled up or shoulder straps fastened. This avoids irritating the ears. If the ears are not injured, trunk tubes may also be donned like a tee shirt (ie, pulled over the head).

The fit can be checked by how the garment feels to the casualty and how the scars feel to the experienced therapist, through the garment. The garment should be tight enough that it is not possible to grab hold of it easily and pull it away from the skin. Shoulders, elbows, and knees should have adequate relief for full AROM without causing open areas. Listen closely to any complaints from the casualty and evaluate them accurately. The garment should not have wrinkles in it. Many times the first garment does not fit correctly. If the garment fits well, an additional set is provided. If it is not a good fit alterations can be made or new measurements taken. Most commercial companies will replace a problematic garment within the first 7 days.

The garment is worn 23 hours a day throughout the duration of the skin maturation process. The garment must be removed for bathing, and occasionally it is necessary to remove it during vigorous exercise sessions when it is causing blisters. It should be washed daily to remove perspiration, body oils, and dirt. Meticulous hygiene is essential for the skin, as well. The garments should be cared for as the manufacturer recommends. Some are hand washable and air dried; others are machine washable.

Some helpful tips may make a difference. A very light cornstarch or powder dusting may help with garment application. Remind the casualty to wash the powder away each day to avoid a plugged pore condition. Wearing nylon under the garment can decrease the shear on skin and increase the ease of application. Foam pads at the joint creases of the knee and ankle can prevent the garment from cutting into the underlying skin. A Duoderm protective patch on the olecranon, the antecubital area, or both, can prevent scrapes in these vulnerable spots. Plastic food wrap over lotion on dry, scaly areas can keep moisture in and protect the garment. In the summer, when the skin is prone to heat rash, the garment can be worn damp to keep the person cool. Some laborers carry a cooler of ice frozen in a sprinkle bottle to cool themselves at work. Old garments are used for swimming.

Reassessment is best done weekly initially, to assure wearing tolerance and to watch for any complications and changes in weight or muscle mass. If an aggressive therapy program is not required, the scars are becoming soft and light in color, and the patient is doing well, visits can be decreased to biweekly, monthly, and bimonthly through the maturation process. Generally, prefabricated garments last only 1 to 2 months, depending on the patient's activity level. A new garment should be issued at each clinic visit until the patient has five garments that fit well. Custom garments generally last 2 to 3 months. Some patients may require more than 2 sets, secondary to their work or leisure situation. It may be necessary to set aside one set of garments for dress and use the stained ones for daily activities. On return to the clinic setting, the state of the skin (contractures or hypertrophic scar tissue) is compared with the previous visit. Narrative or photographic comparison should be made. An objective score can be obtained through measurements based on the assessment form developed by Vancouver General Hospital[137] (Figure 10-96).

If there are bothersome open areas, they should be checked for infection. If they interfere with motion or cause excessive pain the garment can be removed for exercise. It may be necessary to wear Ace bandages for a few days before reapplying the garments. Hands and feet should be checked for a narrowing of the transverse arch. The burn team must remain alert for delayed growth in children who are wearing elastic pressure garments for a long period of time.[126] Parents are also taught to check for regression of skeletal growth. The garment is modified in response to any complications, and reordered when it is fitting well, but the elastic is worn out. Remeasuring is necessary if there has been a significant weight change or growth, as in the case of a child.

The wound is mature when the skin texture is soft and scars appear of proper color and are flat and thin. The skin will be more flexible with some extensibility. There may be some loose, excess skin folds appearing to be wrinkled. Erythema will have faded or lightened from a purple color to red, to pink, to dark brown or white. In people of color, the skin will return closer to its normal pigmentation. Most of the time the pattern of a mesh graft can be seen. Even with optimal results, there is usually some change in color tone. The external vascular supports decrease the amount of hyperpigmen-

GUIDELINES FOR USING THE VGH BURN SCAR ASSESSMENT

This document is intended to clarify procedures for selection of a scar and to offer guidelines for using the VGH Burn Scar Assessment Form. The form is composed of 3 sections: body diagram, rating scales, data record

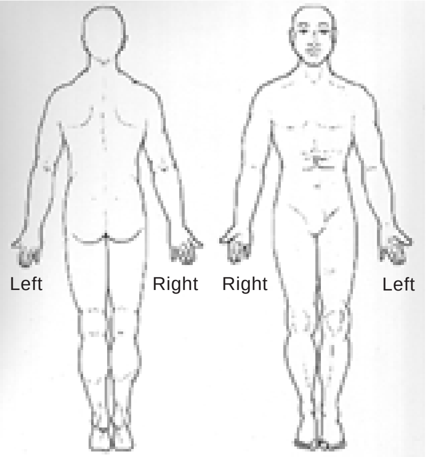
1. SELECTION OF SCAR

Prior to using the VGH assessment, pressure garments should be removed for a minimum of 5 minutes and the area should be in a non-dependent position.

One or more scars are selected at the rater's discretion. As a guide, it is recommended that selection be based on the scars' potential to limit joint range, to become excessively hypertrophic or to require specific therapeutic intervention. If possible, choose the perimeter of the scar to permit greater accuracy for comparison with the normal skin. The area of any scar being rated should not exceed 25 mm x 25 mm (l " x l "). It is recommended that one form per scar is used to allow easy monitoring of scar maturation.

2. BODY DIAGRAM

Once selected, the location of the scar is circled on the schematic body diagram and a number is written adjacent to it. As an adjunct, Polaroid photos may be used, encircled and numbered in a similar manner.



3. RATING SCALES

In the analysis of each scar, 4 components are assessed: pigmentation, vascularity, pliability and height. Each component has a separate scale with zero as the normal reference point.

PIGMENTATION

This is assessed by applying pressure with a piece of clear plastic (for example, 'UVEX') to blanch the scar. This eliminates the influence of vascularity, so that a more accurate assessment of pigmentation can be made. The blanched scar is compared to a nearby blanched area of the person's unburned skin. A variation from the normal skin colour indicates a pigment change. Scale ratings are as follows:

0 - normal (minimal variation from the normal skin pigmentation)
1 - hypopigmentation
2 - mixed pigmentation
3 - hyperpigmentation

VASCULARITY

This is assessed by observing the colour of the scar at rest. In addition, the scar is blanched with the clear plastic and the rate and amount of blood return are observed The more intense the colour return, the higher the rating. Scars which are congested and refill slowly or cannot be completely blanched are grouped in the purple category.

0 - normal (colour and rate of its return, closely resembles that of normal skin)
1 - pink
2 - red
3 - purple

PLIABILITY

This is assessed in the following manner. The scar is positioned to minimize its tension, after which it is manually palpated between thumb and index finger to assess how easily it distorts under this pressure.

0 - normal (resembles pliability of normal skin)
1 - supple (flexible with minimal resistance)
2 - yielding (can be distorted under pressure without moving as a single unit, but offers moderate resistance)
3 - firm (inflexible; scar moves as single unit)
4 - banding (rope-like tissue that blanches with extension of the scar; full range of movement)
5 - contracture (permanent shortening of scar producing limited range of movement)

FIGURE 10-96, Guidelines for Scar Assessment, *continues*

HEIGHT

The height of the scar is visually estimated to be the maximum vertical elevation of the scar above the normal skin. A millimetre scale is included on the form to facilitate this assessment.

0 - normal - flat, flush with normal skin.
1 - > 0 to 1 mm (more than one quarter of the area being rated is raised more than 0, but less than 1 mm)
2 - > 1 to 2 mm (more than one quarter of the area being rated is raised more than 1 mm, but less than 2 mm)
3 - > 2 to 4 mm (more than one quarter of the area being rated is raised more than 2 mm, but less than 4 mm)
4 - > 4 mm (more than one quarter of the area being rated is raised >4 mm)

VANCOUVER GENERAL HOSPITAL
OCCUPATIONAL THERAPY DEPARTMENT

PATIENT NAME:

PIGMENTATION (M)

0 normal - colour that closely resembles the colour over the BURN SCAR ASSESSMENT rest of one's body.
1 hypopigmentation
2 mixed pigmentation
3 hyperpigmentation

VASCULARITY (V)

0 normal - colour that closely resembles the colour over the rest of one's body.
1 pink
2 red
3 purple

PLIABILITY (P)

0 normal supple - flexible with minimal resistance
2 yielding - giving way to pressure
3 firm - inflexible, not easily moved, resistant to manual pressure
4 banding - rope-like tissue that blanches with extension of scar
5 contracture - permanent shortening of scar producing deformity or distortion

HEIGHT (H)

0 normal - flat
1 > 0 to 1 mm
2 > 1 to 2 mm Scale in mm
3 > 2 to 4 mm
4 > 4 mm

Fig 10-96. Vancouver General Hospital Burn Scar Assessment. Based on the assessment form developed by Vancouver General Hospital, 855 West 12th Avenue, Vancouver, BC, Canada V5Z 1M9

tation deposited in the wounds of people of color (Figure 10-97).

When a scar appears to be mature, a trial period without external vascular support is initiated for at least 1 to 3 days but not longer than 1 to 2 weeks. If the symptoms or signs of active scar formation do not recur, the ultimate outcome has been achieved. In evaluating the scar maturity, some professionals consider the scar inactive when less than 10% of the wound remains immature or hyperemic. However, the small hypertrophic area often is more bothersome to the casualty than the large one when it becomes painful, pruritic, and fragile. Continuing use of vascular support garments can prevent this complication.

For a list of prefabricated and custom garment companies,[120] see Exhibit 10-5.

An insert is used to accelerate the hypertrophic scar maturation process.[65] It fills in concave areas that a custom measured garment cannot reach. It is usually placed between the skin and the compression garment over a thick hypertrophic scar, tight

EXHIBIT 10-5

COMPANY PRODUCT AND SERVICES AVAILABLE

This list of products and services is provided here for information only. The mention of these products and services here is not to be construed as an endorsement by the U.S. Department of the Defense, the U.S. Department of the Army, or the US Government.

Prefabricated Garments: Acme United Corp. (800)-243-9852 Note: Tubigrip products were formerly handled by Sepro in the USA and Seton in England. Cloth and paper measuring tapes, measuring guidelines and garment charts provided. Regional representatives supply in-house teaching with a network of local suppliers. Phone discussions are welcomed.

Caromed Int'l., Inc. (800)-833-2237 Polli surgical garments use regular measuring tapes if i.e., circumferential measurements are needed (i.e., a waist size), but some garments vary with Velcro. Sizes by s-m-l. Standard size vests 36", 42" and bra cup size. Order from two national distributors. Phone discussions are welcomed.

Aris Isotoner, Inc. (800)-223-2218 Isotoner gloves are one size fits all. There are some variations in s-m-l corresponding with standard glove sizes 6 to 8 ½. They are variable lengths. Order forms are available. They are also found in some medical catalogues or local stores. Phone calls are welcomed.

Elastic Sport Garments: Bike compression support performance shorts, (92.5% polyester/3.5% Lycra-Spandex), s-m-l-xl, price $15 each, 2801 Redog Drive, Knoxville, TN 37914, phone 1-800-272-6060. This fabric gives protection, and is supportive. Patients often wear the shorts reverse side out to avoid wound abrasion from the seams. Wilson Compression Shorts, 4-way stretch sports girdle, (75% Nylon/25% Lycra-Spandex), s-m-l-xl, price $14.99 each, Martin Manufacturing Co., Inc., 5200 Highway 153, Piedmont, South Carolina 29673, phone 1-800-237-9356. This fabric gives protection, but is not ideal for support. Patients like the "slippery" feel of the fabric, worn reverse side out, which prevents blisters from the seams. Brawn Weightlifter Short (20% Spandex/80% Nylon with full 100% nylon liner), s-m-l-xl, price $24 each, through International Male Magazine, 7412 "F" Street, San Diego, CA 92112-9027. This fabric gives protection, and if the fit is snug, support. Patients say these shorts are preferable because of the soft liner. Stromgren Supports knee length, 25% Lycra/75% Nylon, price $12 each. This fabric is too thin to give support or protection. It is hot. The legs of the shorts are too short to cover donor areas. Beware of discount prices. Measured in standard sizes at your local biking, aerobics or sporting stores.

Custom Garments: Barton-Carey Medical Products (800)-421-0444 A measuring device, "locator" tapes to use with the measuring device, measurement forms, and order forms are provided at no charge; a measuring instruction booklet can be obtained from the company. On request, Barton-Carey sends qualified personnel to train staff members in any hospital in the United States or Canada; for any hospital preferring not to do the measuring, the company will provide that facility with an outside source to do the measuring. Telephone conversations about measurement problems are invited.

Bio-Concepts, Inc. (800)-421-5647 Measurement tapes, a measurement kit (containing a tape "gun," a transparent flexible ruler and other measuring and marking devices, measurement forms, and order forms) are obtainable at no charge. Periodic training seminars for select qualified personnel are held. Bio-Concepts invites participants to this expense-paid seminar; there were 44 seminars with 261 participants as of 1990. A free videotape for patient education is available. Problem solving is encouraged and can be done by telephone. Prefabricated garments are available.

Gottfried Medical, Inc. (800)-537-1968 Paper measurement tapes and measuring forms that use a cloth measuring tape are supplied at no cost. A 20-minute instructional video on measuring and fitting techniques can be provided. The company furnishes support to facilities that request measuring services through an extensive dealer network. Telephone dialogue regarding any questions or difficulties about specific measurements of a pressure support is encouraged.

Jobst Institute, Inc. (800)-537-1063 Paper measuring tapes, measuring forms, and order forms are available at no cost. Training opportunities include a measuring and fitting manual, a measuring and fitting video. 32 service centers in the United States and Canada with staff trained to measure and fit as well as to teach the techniques, five regional service managers available for in-house teaching of the procedures, and a network of dealers throughout the United States and Canada who can serve patients living great distances from the burn center. Phone discussions about orders are welcomed. Prefabricated and interim garments are available.

Wright Linear Pump, Inc. (800)-631-9535; Sticky back measuring tape, measuring forms and Rx forms (800)-922-4226. Forms are available at no charge in a measuring kit. The measuring forms are self-explanatory. There are consultants that use this product in hospitals and training centers across the country. Telephone discussions are welcomed. Note: This is for extremities ONLY that are edematous and used in conjunction with the pump.

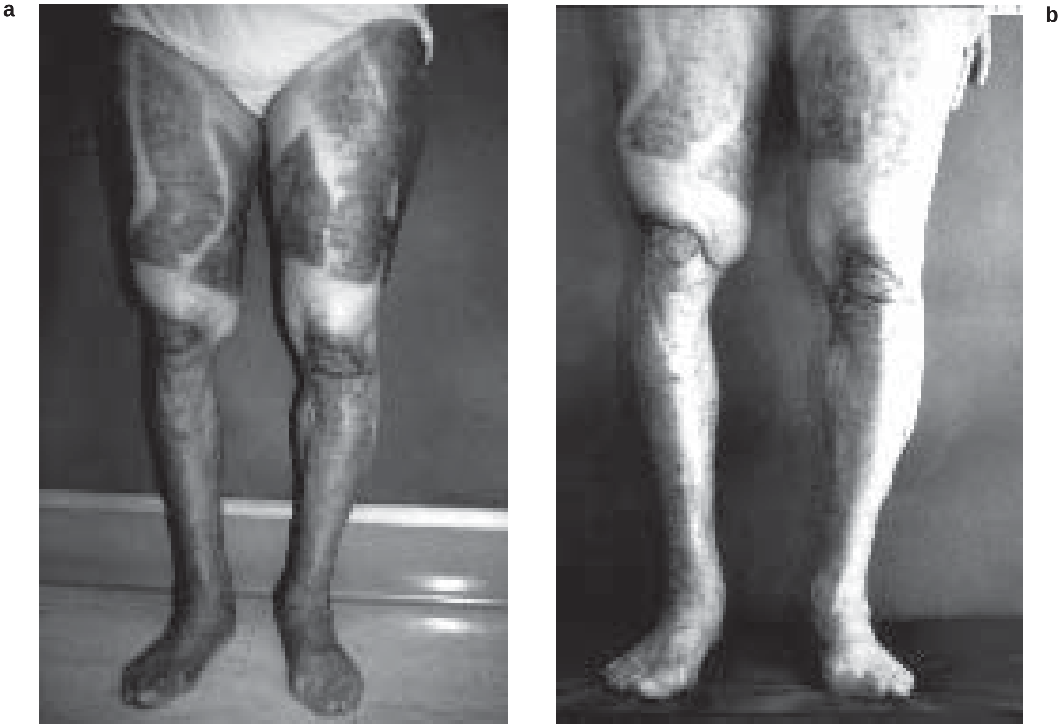


Fig. 10-97. Outcome of deep thermal injury. (**a**) Impaired venous return, dependent immature graft and donor. (**b**) Mature graft and donor; note the permanent cosmetic changes secondary to excision of fascia.

skin, contracture, or a concavity where a bridging area has begun. Some inserts have been used successfully alone, for small areas, without compression. Overlays are placed over the custom measured garment with an elastic wrap or another garment to press the garment into concavities in between bony prominences or anatomical structures that compression garments "tent" over. The overlay or insert fills in the negative spaces for a smooth total contact compression from the external vascular support garment. Inserts also are used to help flatten and soften taut, rope-like contractures. These are often noted over joints. Eventually the tissues become longer, so that blanching is decreased when the skin is put on a stretch, and contractures decrease. Inserts add extra pressure over thick nodular hyperemic scars. When used in this manner, tapering the edges of the inserts will assure total contact by preventing tenting from the elevated ridges. Inserts can be used as padding for protection over bony prominences, under zippers, and at the inner angle of flexor creases, such as the ankles or elbows, to prevent the garments from cutting into the skin. Inserts and overlays help elongate, flatten, and soften scar tissue. Closed cell inserts may need to be worn with an open cell, fabric or disposable gauze, or paper liner to absorb perspiration. A pocket can be sewn into the compression garment to hold inserts (see Figure 10-85). They can also be held in place by applying Velcro to the insert and the garment or running a Velcro strap through the insert itself (Figure 10-98).

Wounds must be healed prior to the use of inserts. Inserts can be fabricated prophylactically for areas of anatomical vulnerability, such as the thumb web space, or concave areas, such as between the breasts (Exhibit 10-6). At the onset of the skin shrinking before contracture can form, a properly placed insert prevents more difficult problems later. As the dynamic scars change, inserts can be serially made for increasingly larger anatomical spaces as the skin stretches. As the scar flattens, impressions can be filled in until the insert is flush with the surface and the scar impression is no longer seen in the insert. Eventually, during the course of maturation, inserts can be made completely flat, using a rolling pin. Larger inserts can be made flat on old x-ray films. Reevaluation and modifications are made at regular outpatient visits.

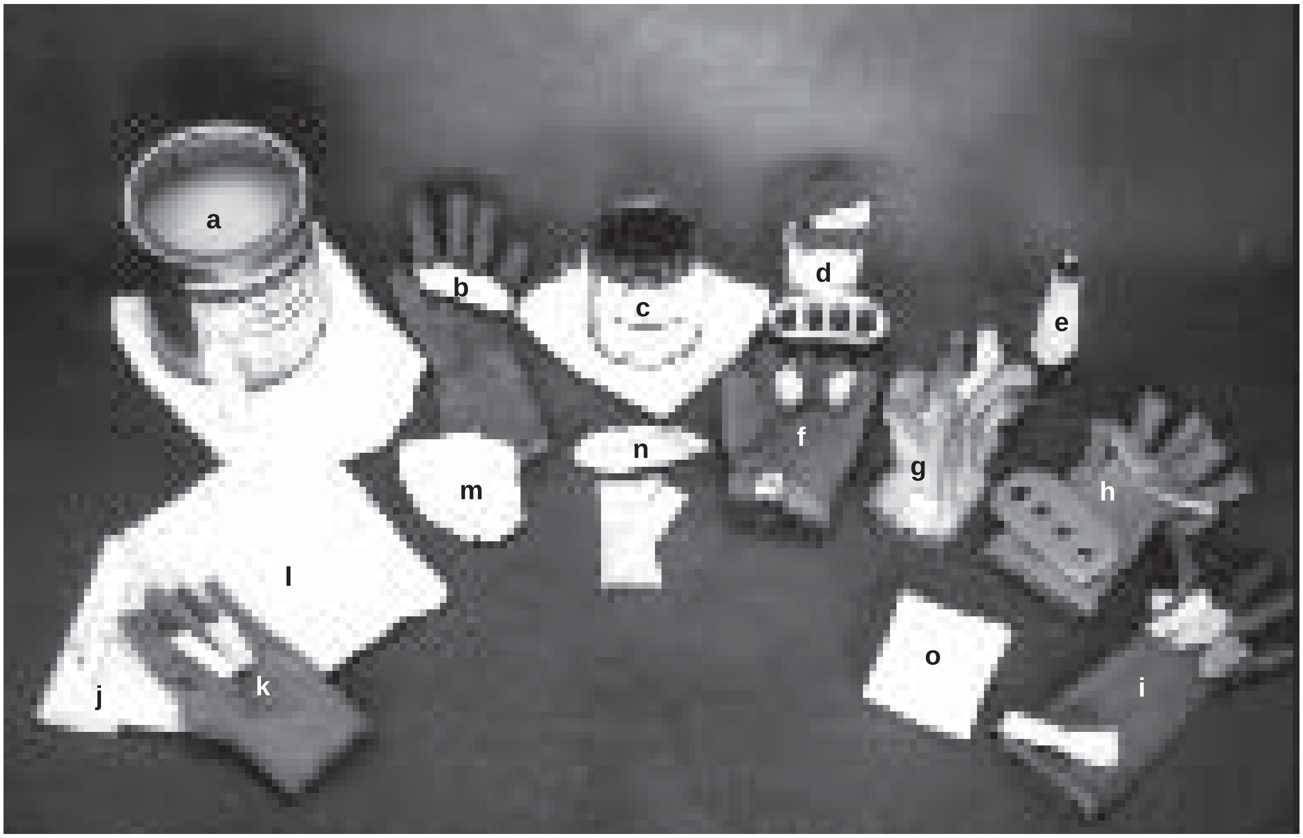


Fig. 10-98. Examples of materials for inserts, overlays, and accompanying support gloves: (**a**) Rolyan Silicone Elastomer; (**b**) Isotoner glove and silastic elastomer "brass knuckles"; (**c**) Smith and Nephew Prosthetic foam material; (**d**) Otoform K material and Otoform K "brass knuckles"; (**e**) prosthetic foam palmar insert; (**f**) prefabricated Jobst glove with Otoform K web spacers; (**g**) Jobst interim glove with stockinet overlay (note applicator distal to fourth finger); (**h**) prefabricated Tubigrip glove with Velfoam "brass knuckles"; (**i**) Isotoner glove with finger inserts (left to right—orthopedic felt, lamb's wool, Betapile, at wrist: small orthopedic felt split into layers); (**j**) Elastogel cast and splint pad; (**k**) Bioconcepts prefabricated glove with Otoform K separated interdigital web inserts; (**l**) Spenco skin care pad; (**m**) prosthetic foam palmar insert, prosthetic foam palmar insert; (**n**) Jobst interim pediatric gauntlet; (**o**) orthopedic felt layers.

There is a wide variety of materials to use for inserts or overlays to increase pressure over a hard scar. The creativity of both therapists and casualties has been the source of many successful scar modifiers. Fabric, open or closed cell foam, rubber or plastic pieces, and silicones are all appropriate inserts or overlays (Figure 10-99). Inserts can be ready made and cut to the individual's needs, or they can be custom formed to the body's contours. These inserts can be secured in external vascular support garments by pockets (Figure 10-100). Fabric envelopes, Webril (Kendall Healthcare Products Co.), or other disposable wrap may be used to protect the skin from direct contact with the oily silicone. Often the silicone can be placed between two Isotoner gloves or between two Tubigrip stockings, which increases the pressure on the scar and prolongs the life of the insert. When contact dermatitis is caused by the insert (Figure 10-101), that insert must be discontinued and after the area is healed a different insert material tried.

The insert and support must be applied in a way to maintain gradient pressure. They cannot be applied like a tourniquet in the middle of an extremity. This will impair venous return and cause edema in the distal hand or foot. Even if edema is not severe enough to be seen with the naked eye, the distal circulation will change. Therefore, the insert must be placed under or over a support that is donned from the hand or foot to the shoulder, knee, or hip. The following prefabricated commercially available fabrics and foams are listed in order of

EXHIBIT 10-6

INDICATIONS, CONTRAINDICATIONS, AND PRECAUTIONS FOR USE OF INSERTS WITH EXTERNAL VASCULAR SUPPORTS

Indications
- Area of concavity (ie, chest sternum, palmar arch)
- Particularly active hypertrophic scar
- Contracture bands
- Use in both old and new scars
- Problematic body areas vulnerable to contracture (ie, finger webs, popliteal, and antecubital fossae)
- Bony prominences vulnerable to breakdown

Contraindications
- Sensitive skin or prior dermatologic condition such as prolonged use of steroid creams
- Prior skin diseases (ie, eczema)
- Impaired cognition
- Open wounds
- Allergies

Precautions
- Maceration secondary to moisture build-up
- Friction blisters
- Decubiti
- Contact dermatitis
- Heat rash
- Allergic reactions

increasing abrasiveness. Lamb's wool in coil form is a very soft natural fiber. It absorbs moisture in interdigital web spaces and protects fragile areas. It is useful with people fearful of replacing their present dressings with another texture. Fibers will adhere and incorporate into open wounds and therefore wool should be covered if applied where open areas or drainage is present.

Orthopedic felt is a white polyester fabric padding ⅛, ¼, or ½ in. thick that can be cut into various sizes and shapes. It can be separated into layers to vary the thickness. The inner surface is softer. It thins out with repetitive use and must be replaced often.

Sheepskin is a thick, fluffy synthetic material that looks like real sheepskin with a firm fabric backing. It is good for protection of the bony prominences or for increasing compression.

Moleskin is a very thin soft padding fabric with self-adhesive backing. It can be placed against garments or splints for protection and to even out two surfaces.

Hypafix is a contact medium in the form of a soft adhesive gauze. It can be left in place for several days and creates tension on anatomical depressions. Success has been reported using this in place of compression garments over small areas.[138]

Velfoam and Betapile are cushioned strapping materials that work well as finger inserts. They are loose fabric compatible with hook Velcro.

Hollister Odor Absorbent dressing is a thin, absorbent, open cell, charcoal black foam encapsulated by a soft white mesh material. It is machine washable and dryable. It works very well as a liner to absorb perspiration or prevent maceration under elastomer inserts.

Reston foam is a white, open cell foam. It compresses with pressure. The adhesive adheres to wood or plastic securely and cannot be removed easily. The foam also absorbs odor and can be unpleasant in the presence of drainage.

Polycushion is a beige, closed cell foam with an adhesive back. It can be placed on splints or gar-

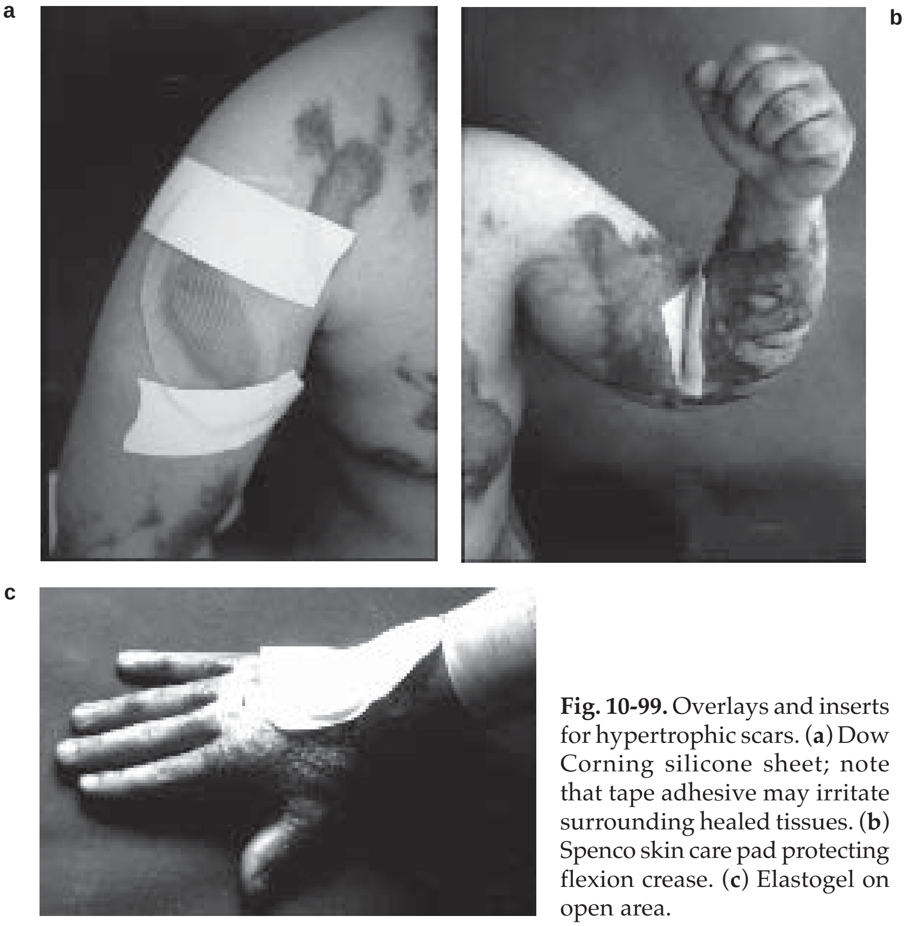


Fig. 10-99. Overlays and inserts for hypertrophic scars. (**a**) Dow Corning silicone sheet; note that tape adhesive may irritate surrounding healed tissues. (**b**) Spenco skin care pad protecting flexion crease. (**c**) Elastogel on open area.

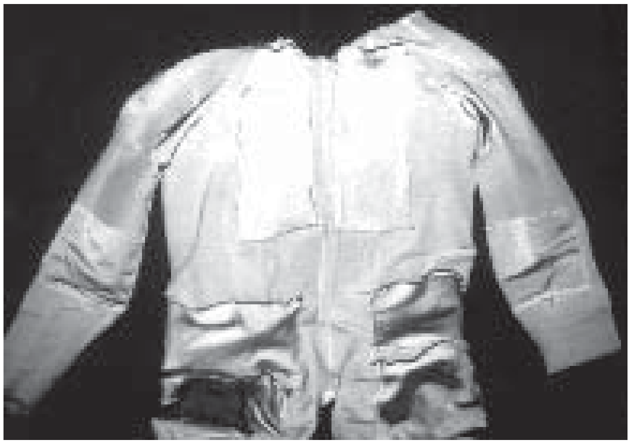


Fig. 10-100. Custom measured Barton Carey garments. The garment is turned inside out to show the pockets that secure the silicone inserts.

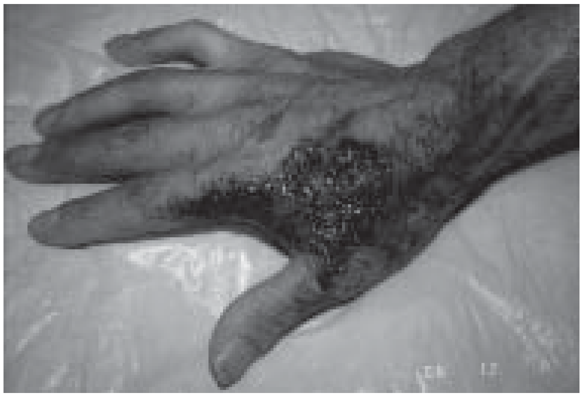


Fig. 10-101. Contact dermatitis caused by the Otoform K thumb insert.

ments. The two adhesive sides can be folded together to make a nonadhesive insert.

Kushion flex is a white, very firm, closed cell padding with adhesive backing. It may be beneficial in an area where durability is required such as a shoe insert.

Plastazote is a flesh colored, closed cell foam that can be formed on the skin after it is heated to a low temperature. It is also good for shoe inserts. It comes in either adhesive or nonadhesive forms.

Neoprene is a blue-black, closed cell rubbery material. It is spongy with elastic memory and is available in variable thicknesses.

Contour form is a blue colored, open cell, slow recovery padding. Body heat helps to mold the foam. It is available in different thicknesses.

Microfoam tape is a soft tape similar to Reston but of closed cell foam. The two adhesive sides may be placed together to form an insert or zipper pad.

The following silicones and elastomers are also listed according to increasing abrasiveness:

Skin care pad (formerly known as Dermapad) comes in 4 x 4 in. by ⅛ or ½ in. sizes. It is a cream colored protective pad with a solid gelatinous consistency described as artificial fat. It is lightly adherent and cuts into any shape. It provides cooling comfort. Thicker pads eliminate friction and absorb pressure. Thinner, flexible pads are excellent for use on mobile joints such as the elbow. Both sizes are excellent to increase compression. The pad's surface is very oily, and the scar must be protected or observed for maceration. Using these pads for 8- to 12-hour periods, and leaving them off for 12 to 16 hours may relieve skin over-moisturization.

Dow Corning silicone gel sheeting (see Figure 10-99) is a clear, soft, slightly adherent, semiocclusive, flexible insert made from medical-grade silicone polymers without fillers.[139,140] One version is very tacky, stays in place well, but crumbles fairly easily. Another type has a netting back woven through. It is unique in that it is advertised to modify a scar with or without pressure. When it is effective, it is worn a minimum of 12 hours per day rather than the usual 23 hours per day.[141,142] If both compression and the silicone gel sheeting are worn, they should only be worn together 12 hours a day. For the other 11 hours, one or the other can be worn separately. Otherwise, skin irritation may develop and the gel sheet will have too much wear, disintegrating it.

Otoform K ([Dimethylpolysiloxane] Dreve Co.) is a white silicone elastomer with a putty base. It is mixed with a red tube catalyst resulting in a firm, pink, rubbery, closed cell insert. It does not run, sets in five minutes, is odor free and semirigid but flexible. It positions, yet allows some motion. It is easy to work with in small to medium quantities. It works well for interdigital web spacers of the hand, thumb, and feet, and as a shock absorber, decreasing contractures and filling in concavities (see Figure 10-72).

Silastic (Dow Corning) elastomer is a closed cell, gray-liquid-based, taffy-like elastomer mixed with a clear catalyst out of an eye dropper. It cures to a rubbery texture. The setting rate depends on the amount of catalyst used. It is a little more challenging to handle, requiring a tongue depressor. It sticks to hair, so a protective layer on the skin of lotion or plastic wrap is helpful. It does stain before the catalyst is added. It requires refrigeration or storage and has a 1-year shelf life. It picks up very fine detail. It is fairly rigid and works well on large areas. Serial inserts can be made by filling in the impression of the scar as it improves with silastic medical adhesive. Repetitive fabrications can be made on a revised scar until it is flush. On large body areas a pattern can be made and the insert fabricated on old radiographic material. It works well on flatter surfaces with less mobility such as the trunk, lateral ankle, or palmar arch. It can be mixed with prosthetic foam.

Dow Corning prosthetic foam is an open cell, tan colored, liquid-based, silicone elastomer mixed with a clear bottle of catalyst. When the catalyst is added, it will increase in volume and have an exothermic reaction. It can be contained by forming it inside of transparent plastic wrap against the body. It sticks to hair. It is soft and spongy. It can be used in conjunction with the hard, low temperature splinting material outer shell. It works well as a total contact padding on delicate or fragile areas. It is sometimes placed on a fresh graft with a moist sterile dressing underneath it.

Elastogel (South West Tech, Inc.) cast and splint pads can be used as an insert under a compression garment (see Figure 10-99c). It has a nice feature of a cloth covering on the back, protecting the garment from its oils. It is also an occlusive dressing that can be used on open wounds. The insert can be cut to size, but should extend 1 to 2 in. beyond the wound or be 25% larger than the wound. It can be worn over topical medication; it can be made waterproof by covering it with a film dressing such as Tegaderm (3M Health Care); and can be secured with tape, gauze, or elastic compression.

Spenco (Spenco Medical Corp.) second skin is a clear, breathable-type, hydrogel gel. It is 96% water and 4% polyethylene oxide with a feel and consistency similar to human skin. It removes the fric-

EXHIBIT 10-7

PRIORITIZED OPTIONS FOR SCAR COMPRESSION—UPPER EXTREMITY

Fingers-External vascular support must be satisfactory at the most distal part (fingers or hand) to prevent a tourniquet effect if any support is planned for the wrist, forearm, elbow or upper arm.

- I. Compression Garments
  - A. Finger wraps
    - 1. Sof-Kling, one inch rolls, used in spiral wrap
    - 2. Unna bandage, split, folded and wrapped in a spiral, distal to proximal
  - B. External vascular support, Tubiton finger oedema sleeve, digisleeve
  - C. Isotoner glove
    - 1. Elastic spiral wraps (Elset, Coban, PEG self-adhesive bandage, regular elastic bandages, 1- or 2-inch size)
    - 2. Elastic, old fashioned “boxer” type, finger and hand figure-8 wraps (Must have adequate strength to overcome dorsal pull of wrap, or wrap must be applied in reverse “boxer” style.)
  - D. Silastic mitt or foam “sandwich” secured and supported with elastic wraps
- II. Inserts
  - A. Finger Webs
    - 1. Web spaces
      - a. When fragile:
        - (1) Lamb’s wool strips
        - (2) Webril strips in rolls
        - (3) Orthopedic felt strips
        - (4) Hypafix strips
        - (5) Velfoam or Betapile strips, formed in “brass knuckles” pattern
        - (6) Various foams
      - b. When durable:
        - (1) Otoform K “saddle” pattern with Webril wrap to prevent contact dermatitis
        - (2) Silastic elastomer “brass knuckle” cut-out pattern
        - (3) Skin care pad or Elastogel strips
        - (4) Plastazote strips
        - (5) Neoprene strips or cutout in “brass knuckle” pattern
        - (6) Various foams strips
        - (7) Thermoplastic inserts
  - B. Fingers
    - 1. Volar surface
      - a. When skin begins to contract:
        - (1) Otoform K “gutter” secured with one inch Sof-Kling or Coban or equivalent
        - (2) Silastic elastomer thin strip under glove
        - (3) Skin care pad strip
        - (4) Elastogel strip
        - (5) Thermoplastic “gutter” splint from the tip of finger to the “V” of the digit or palmar base

Thumb

- I. Compression Garments
  - A. Sof-Kling, one inch rolls, used in figure-8 wrap thumb to wrist, to abduct thumb
  - B. Unna bandage, split, folded, and wrapped in a spiral, distal to proximal
  - C. Elastic wrap including the thumb, hand, and fingers
  - D. Prefabricated or custom gauntlet including thumb hand and base of index finger
  - E. Prefabricated or custom glove
- II. Inserts
  - A. Thumb Web
    - 1. Orthopedic felt “saddle” shaped pad
    - 2. Webril “saddle” pad
    - 3. Velfoam or Betapile “saddle” pad
    - 4. Skin care pad
    - 5. Elastogel

EXHIBIT 10-7 *continues*

EXHIBIT 10-7 *(continued)*

PRIORITIZED OPTIONS FOR SCAR COMPRESSION—UPPER EXTREMITY

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6. Otoform K saddle to thumb IP and index MCP
7. Sheepskin
8. Various fabric liners

B. Radial Border
1. Otoform K strip
2. Skin care pad
3. Elastogel

Hand

I. Compression Garments
    A. Isotoner glove
    B. Sof-Kling one inch rolls
    C. Elastic wraps (Elset, Coban, PEG self-adhesive bandage, regular elastic bandages, 1- or 2-inch size)
    D. Unna bandage
    E. Prefabricated or custom gauntlets
    F. Prefabricated or custom gloves
    G. Silastic elastomer or foam mitt
II. Inserts
    A. Palm
        1. Otoform K
        2. Silastic elastomer, sculpted
        3. Skin care pad
        4. Elastogel
        5. Various fabrics as liners, secondary to perspiration
    B. Dorsum
        1. Skin care pad
        2. Elastogel
        3. Second skin
        4. Duoderm
        5. Silicone gel sheet with or without compression

Wrist

I. Compression Garments
    A. Prefabricated or custom long gloves, long sleeves, both overlapping or separate wrist band
II. Inserts—same as elbow

Elbow

I. Compression Garments
    A. Prefabricated, shaped support bandage or tube; prefabricated single arm, custom sewed shaped support bandage with larger elbow insert, prefabricated or custom measured long sleeve vest
    B. Custom measured arm sleeve
    C. Antecubital or circumferential liner (Note liner fabric is tighter than the elastic garment and may cause irritation if not relieved. Shearling inserts may be used for extremely fragile areas.)
II. Inserts
    A. Anterior
        1. Skin care pad
        2. Elastogel
        3. Spenco second skin
        4. Duoderm
        5. Moleskin
        6. Hypafix
        7. Hollister odor absorbing dressing
        8. Various fabrics
        9. Various foams

EXHHIBIT 10-7 *continues*

EXHIBIT 10-7 *(continued)*

PRIORITIZED OPTIONS FOR SCAR COMPRESSION—UPPER EXTREMITY

- B. Posterior
    - 1. Duoderm
    - 2. Foam sponge
    - 3. Various pads

Upper Arm and Forearm

- I. Compression Garments—Same as elbow
- II. Inserts
    - A. Dow Corning silicone gel sheeting with or without compression
    - B. Silastic elastomer
    - C. Otoform K
    - D. Adhesive backed foams
    - E. Skin care pads
    - F. Elastogel
    - G. Neoprene

Shoulders

- I. Compression Garments
    - A. Prefabricated, interim or customized vest with long sleeves
    - B. Vest with short sleeves
    - C. Axilla can have netting if it is unburned. Axilla may be left open if durable and nonburned
    - D. Figure-8 Ace wrap with foam inserts
    - E. Clavicle straps
- II. Inserts
    - A. Axilla
        - 1. Skin care pad
        - 2. Elastogel
        - 3. Prosthetic foam/elastomer mix
        - 4. Various thick foam paddings
    - B. Superior Shoulder
        - 1. Skin care pad
        - 2. Elastogel
        - 3. Various thin adhesive-backed back foam padding
        - 4. Spenco second skin
        - 5. Otoform K
        - 6. Duoderm, if friction is a problem
        - 7. Silicone gel sheet, if shoulder motion does not displace it

tion between two moving surfaces and will not stick to wounds or dressings. It keeps tendons hydrated and cushions blisters, protecting and preventing them. It helps ease burn pain and itching; it is placed directly against the skin and it is held in place with a bandage or tape. The outer plastic film can be left in place to prevent drying or removed to allow air passage; uncovered, it will dehydrate after 24 hours, but placing it in water will return it to its original position.

Duoderm is a thin, caramel colored, hydroactive, cell-adherent occlusive dressing. It works well on bony prominences for protection and can be used over mobile joints such as the posterior elbow by cutting darts into it and holding it in place during flexion, melding it into position. It works well on hands and allows a tight compression glove to be applied over it.

There are many different ways to provide effective compression to body parts that have scars for various other types of problems. Exhibits 10-7, 10-8, and 10-9 provide lists of prioritized options for specific situations in regard to scar compression using external vascular support garments and compression inserts or overlays.

In cases where grafts are onto fascia or a face develops poor cosmetic outcome, camouflage makeup is indicated. Additionally, accessories can

EXHIBIT 10-8

PRIORITIZED OPTIONS FOR SCAR COMPRESSION—TRUNK

Anterior Flank and Intrascapular Area

- I. Compression Garments
  - A. Prefabricated tube
  - B. Prefabricated or custom vest with or without sleeves
  - C. Female vest with breast cups
  - D. Inserts
- II. Flank and Intrascapular
  - A. Silicone gel sheeting with or without compression
  - B. Silastic elastomer
  - C. Otoform K
  - D. Prosthetic foam
  - E. Skin care pad
  - F. Elastogel
  - G. Spenco II skin
  - H. Various adhesive backed foam
- III. Between Breast Cleavage
  - A. Otoform K
  - B. Prosthetic foam
  - C. Thick sculptured foam
  - D. Silastic elastomer
  - E. Plastazote
  - F. Skin care pad
  - G. Elastogel
  - H. Spenco II Skin
- IV. Buttock/Groin Compression
  - A. Body suit
  - B. Vest with Velcro crotch
  - C. Pants or shorts
  - D. Scrotum may require support cup

Inserts

- I. Buttock Crease
  - A. Otoform K
  - B. Prosthetic foam
  - C. Thick sculptured foam
  - D. Plastazote
  - E. Skin care pad
  - F. Elastogel
- II. Genitalia
  - A. Males may require a foam roll to stretch the penis over if there is constriction during erection
  - B. Males may require a catheter or penile implant for the above or if the urethra is restricted producing difficulty with ejaculation and/or urination
  - C. Female genitalia is usually well protected secondary to anatomical makeup

be used to camouflage, such as wigs, scarves, fashion gloves, adaptive clothing, and prosthetic foam covers, may be needed. If a multidisciplinary approach is used with the soldier as the active principal team member, the outcome can be cosmetically and functionally acceptable. Protection from sunburn is an additional benefit from camouflage make-up applied over the face, ears, nose, and neck.

Functional Activities and Adaptive Equipment During the Wound Maturation Phase

In the wound maturation rehabilitation phase, a casualty continues active participation in functional activities that are consistent with life roles and return to active duty (see Figures 10-62 and 10-102). As an individual becomes medically stable and epi-

EXHIBIT 10-9

PRIORITIZED OPTIONS FOR SCAR COMPRESSION—LOWER EXTREMITY

Thigh and Lower Leg

Same as in the Upper Extremity, upper arm and forearm (Exhibit 10-7)

Hip

- I. Compression Garments
    - A. Biker pants or shorts
    - B. Prefabricated or custom pants or shorts
    - C. Custom measured and fitted body suit with thighs incorporated
- II. Inserts
    - A. Skin care pad
    - B. Elastogel
    - C. Various thin adhesive backed foams

Knee

- I. Compression Garments
    - A. Prefabricated tubes are shaped support bandage or pants
    - B. Custom thigh highs or pants
    - C. Biker pants or long shorts
- II. Inserts
    - A. Anterior knee not usually necessary
    - B. Posterior knee
        - 1. Skin care pad
        - 2. Elastogel
        - 3. Thin fabric liners may be necessary secondary to maturation

Ankle

- I. Compression Garments
    - A. Prefabricated tubes
    - B. Prefabricated or custom knee highs or higher garments
    - C. High anklets
    - D. Boots with elastomer or prosthetic foam circumferential lining
- II. Inserts
    - A. Anterior
        - 1. Foam pad
        - 2. Otoform K
        - 3. Orthopedic felt
        - 4. Skin care pad
        - 5. Elastogel
    - B. Lateral
        - 1. Silastic elastomer
        - 2. Otoform K
        - 3. Foam pad
        - 4. Duoderm
        - 5. Spenco II to skin
        - 6. Skin care pad
        - 7. Elastogel
    - C. Posterior
        - 1. Duoderm
        - 2. Foam pad
        - 3. Silastic elastomer
        - 4. Otoform K
        - 5. Skin care pad
        - 6. Elastogel

EXHIBIT 10-9 *continues*

EXHIBIT 10-9 *continued*

PRIORITIZED OPTIONS FOR SCAR COMPRESSION—LOWER EXTREMITY

Foot

- I. External vascular support
  - A. Prefabricated tube
  - B. Prefabricated anklet or stocking
  - C. Custom measured anklet or stocking
  - D. High top tennis shoe
  - E. Boots with silastic or prosthetic foam circumferential support
- II. Inserts
  - A. Dorsum
    - 1. Otoform K
    - 2. Silastic elastomer
    - 3. Various thin adhesive back foams
    - 4. Skin care pad
    - 5. Elastogel
    - 6. Silicone gel sheet with compression
  - B. Sole
    - 1. Thick foam secured over elastic wrap for comfort
  - C. Ankles and High Top Shoes
    - 1. Sheepskin to protect bony prominences
  - D. Toes that are hyperextended
    - 1. Plantar lift from the heel to the metatarsal head
      - a. Kushion flex
      - b. Polycushion
      - c. Plastazote
      - d. Neoprene
      - e. Silastic elastomer
      - f. Otoform K
    - 2. Plantar negative space for toes to flex into.
      - a. Lamb's wool
      - b. Sheepskin
      - c. Orthopedic felt
      - d. Reston foam
      - e. Contour foam
      - f. Other compressive open cell foams
    - 3. Dorsal insert to flex toes
      - a. Otoform K
      - b. Silastic elastomer with moleskin or odor absorbent liner
      - c. Sheepskin
      - d. Thick closed cell foam

thelium becomes more durable, it is vital to pursue a variety of activities, but, in this phase, it is still important to be cautious with fragile skin. Casualties must learn proper interventions to compensate for sensory, pigmentation, and circulatory changes while performing daily living skills. In this phase of rehabilitation the casualty is weaned off adaptive equipment used in the initial phases of healing. However, electrical injuries, amputees, or casualties whose burns are greater than 70% TBSA may continue to use the equipment if residual deficits exist. In the severely impaired person, sophisticated orthosis and adaptive equipment will need to be pursued to reduce disability.

Feeding. In the wound maturation phase adaptive silverware, drinking aids, and stabilization devices are rarely used. If decreased shoulder AROM with proximal weakness is permanent, suspension slings or mobile arm supports (ball bearing feeders) can be used when a casualty is in a seated position. These devices support the entire arm, use mechanical devices to assist shoulder flexion, and allow the elbow to move in a gravity eliminated plane. The mobile arm support is available

PATIENT'S DAILY SKILLS CHECK LIST

By discharge, all burn patients will have learned to do the following tasks independently:

	YES	NO	Needs more practice	PHN or other relative
I Hygiene/grooming				
Wound Care:				
Wash wound				
Apply medication				
Tub bath				
Shower				
Appropriately wash				
healed and unburned skin				
Lotion and massage				
self appropriately				
Care of overgrafted tissue				
& sweat & oil gland regrowth				
Buff-n-Puff				
Grit Soap				
Shampoo:				
Wash				
Rinse and dry hair				
Trim around open areas				
Comb and brush				
Shave:				
Remove ingrown hairs				
Toileting:				
Approach and sit on stool				
Raise and lower clothing				
Wipe self				
Clean hands				
Oral Hygiene:				
Brush teeth				
Stretch mouth				
Lotion lips				
II. Dressing:				
Independent applying aces				
Independent in bra,				
Independent donning custom (top)				
elastic garment (bottom)				
Independent				
dressing lower body				
including tying shoes				
III. Eating:				
Feed self				
Cut meat, open milk carton,				
sugar pkg., butter, bread				
IV. Homemaking:				
I. Meal Preparation:				
Safe with cold meal				
(milk, sandwich, apple)				
Safe with hot liquids				
(coffee, soup, etc.)				
Safe with stove top and oven				
2. Kitchen Care:				
Wash dishes				
Put away dishes				
Empty dishwasher				

FIGURE 10-102 *continues*

in standard, elevating, and table mounted models. In cases of severe burns resulting in upper extremity amputations, there are battery powered or electric feeding machines that are operated by microswitch control. The switches can be operated by the chin or another body part that can more predictably and consistently control a machine.

Grooming. In the wound maturation phase the casualty can participate in additional grooming tasks, and can perform shaving activities with spe-

FIGURE 10-102 *continued*

	YES	NO	Needs more Practice	PHN or other relative
Sweep floor				
Clean counters				
Clean stove and oven				
Defrost refrigerator				
3. Other:				
Make bed				
Change bed linen				
Vacuum				
Dust				
Wash windows				
Change light bulbs				
Carry and put away groceries				
Do laundry				
Mop floors				
4. Misc.:				
Set and wind clock, watch				
Put coins in machines				
Handle wallet				
Sign name				
Write letter				
5. Clean bathroom				
Change toilet paper roll				
V. Mobility:				
Walk for 10 minutes				
Run				
Stair climbing				
Ride bike				
Safe motor vehicle operation				
Safe riding bus and public transportation				
VI. Recreational Activities:				
1. Knows precautions regarding:				
A. Fragile healed skin				
B. Sensory changes				
C. Pigmentation changes				
D. Circulatory changes				
E. Exposure to irritants, eg. petroleum products, concentrated animal waste				
2. Initiates old or new social contacts				
3. Initiates stress reducing physical recreation 3 times a week or more				
4. Initiates discussion of desires in social interaction				
5. Initiates doing things for others in family or social contacts				
VII. Work Activities:				
Return to old job (usually in 6-12 months)				
Find a new job				
Work with Vocational and Rehabilitation Counselor				
VIII. Return to school:				
Able to concentrate on learning				
Takes part in non-contact sports				
IX. Sexuality:				
Resolving changed body image				
Initiates discussion of desires				

Burn patients who have had grafts to all extremities and the head will routinely be offered intensive therapy in a Rehabilitation Unit and public health nurse assistance to achieve independence at home. The parents or caretakers of all children under 5 years of age will be offered public health nurse assistance. Contact your social worker for help if you are interested in these services.

Fig. 10-102. Clinical history sheet. Reprinted with permission of Regions Hospital (formerly, St. Paul-Ramsey Medical Center), 640 Jackson Street, St. Paul, Minnesota 55101-2595.

cial holders for electric or regular razors if unable to hold a razor in the conventional manner. The casualty should perform nail care to prevent excoriation of skin during scratching. Mounted nail clippers or files are available if needed.

At this stage, the casualty can begin to assume responsibility for the condition of the skin, inspecting it regularly for detection of breakdown areas. Flexible skin-inspection mirrors assist with this. The casualty can also begin to be responsible for independent wound care and management and should demonstrate the ability to safely wash the wounds and to apply medication and gauze dressings. As the skin heals, the casualty should perform scar massage as indicated and apply moisturizer.

In the wound maturation phase a casualty will want to be able to perform toileting tasks independently. There are a variety of types of adaptive equipment to ensure safety and independence with the task. Toilet safety rails can be mounted on either side of the toilet to increase stability with transfers. Use of a raised toilet seat is discouraged, but is useful when an individual permanently lacks strength or control in the lower extremities and cannot perform transfers with a standard seat. A bedside commode is necessary in the hospital or at home only when ambulation to the bathroom will not be possible. Toilet aids are helpful if an individual is lacking the necessary upper extremity range of motion for hygiene.

There are a number of adaptive safety aids to allow independent bathing. Often it is safer for the casualty to bathe in a seated position if endurance is low. Edema is minimized by keeping burned legs elevated until the bathing is finished and external vascular supports are replaced. Shower seats, with or without backs, are useful to provide stability in walk-in showers. Extended tub benches allow a casualty to bathe independently on a seat if he is unable to step over the tub safely. Grab bars can be mounted on the tub or wall to further increase stability with transfers. A flexible shower hose permits a person to bathe independently in a seated position if he is unable to stand for a long period of time. Long handled sponges compensate for decreased trunk or upper extremity range of motion and allow casualties to wash the feet and back independently. A bath mitt stabilizes a bar of soap if the grasp is weak. Nonskid surfaces or bath mats applied to the bottom of the tub are safety features used to avoid slippage.

Dressing. Assuming total responsibility for donning external vascular support garments, orthoses, and clothing is achieved during the wound maturation phase. The casualty must be cautioned against pulling too rigorously on garments while donning them, which could injure fragile skin. A nylon stocking contact layer over gauze dressings secures them and facilitates donning external vascular supports. The casualty demonstrates donning face masks, microstomia splints, hand splints, and lower extremity braces independently in front of a mirror to ensure proper fit.

There are a number of adaptive aids that assist with donning the pressure garments and regular clothing. In prioritizing practice time, often it is more cost effective to practice donning items without adaptations when in 3 or 4 days the trunk and upper and lower extremity contractures will stretch to allow independence without adaptations. Long handled aids are helpful in cases where there is decreased trunk, hip, and knee flexion, and hip external rotation. Many brands of "reachers" can be used to don pants (Figure 10-103). They are available in a standard size, extended length, and a self-closing model for decreased hand grasp. There are long handled shoe horns (Figure 10-104) and stocking aids (Figure 10-105) that are useful in donning shoes and socks. A dressing stick can be used for donning pants or for donning shirts if upper extremity range of motion is limited.

Other adaptive aids are useful if finger dexterity is limited or if a person has use of only one hand. Elastic shoe laces and button and zipper aids are available in standard and easy-grasp varieties.

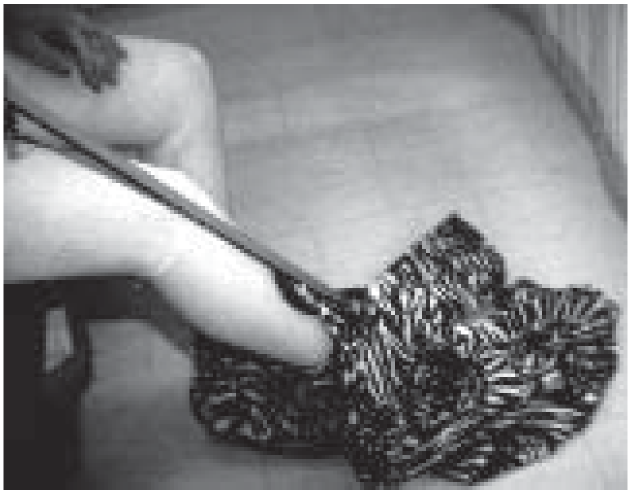

Fig. 10-103. A "reacher" aid for donning pressure garments and regular clothing. The casualty wears Isotoner glove external vascular support when hands will be dependent.

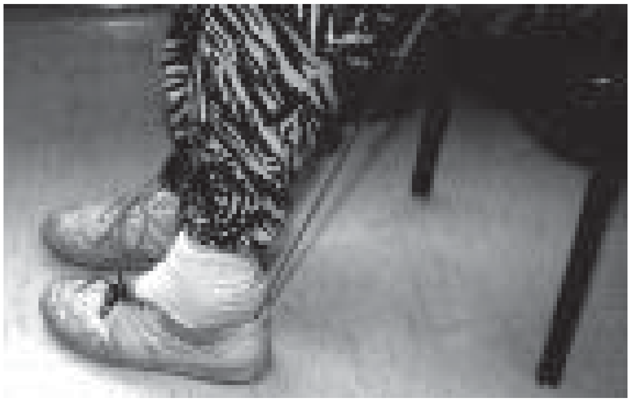

Fig. 10-104. The casualty is using a long-handled shoe horn to facilitate donning shoes.

Velcro closures can replace buttons or zippers, and large loops can be sewn onto pants. In general, some types of clothing are relatively easy to don such as large shirts, gym pants (Figure 10-106), and Velcro closure tennis shoes. There are catalogues for special-order, easy-to-don clothing.

Home Management

As a casualty becomes more independent in the hospital setting, it is important to identify home management responsibilities for eventual discharge. This can involve a variety of tasks including general maneuvering, operating home appliances, cooking, cleaning, and several other activities. It is important for the casualty and family to identify the tasks that the individual wants or needs to perform. Ideally, a survey of home needs can be accomplished with the casualty, family, and a staff

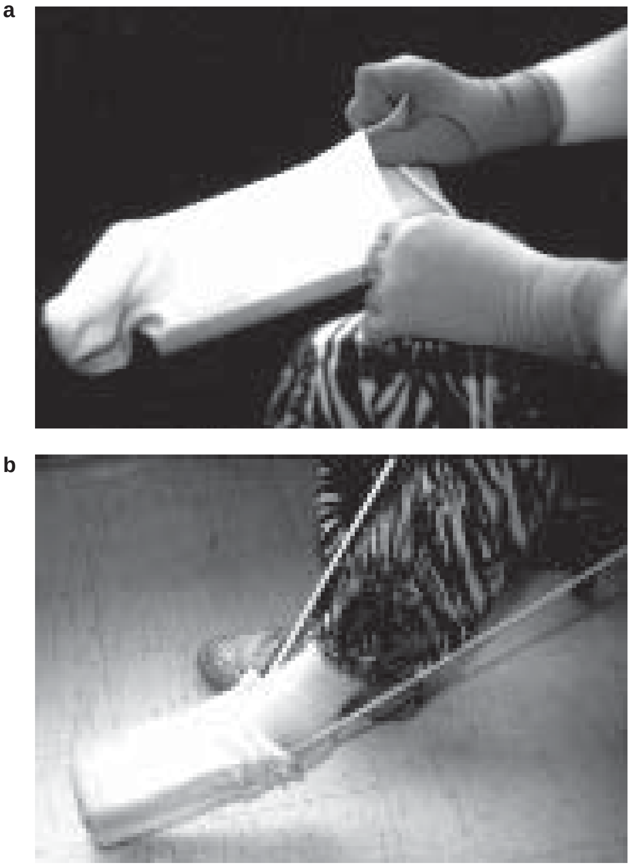

Fig. 10-105. The casualty donning stockings. (**a**) The casualty is placing the stocking over the sock aid and (**b**) is using a sock aid to place the stocking on his foot.

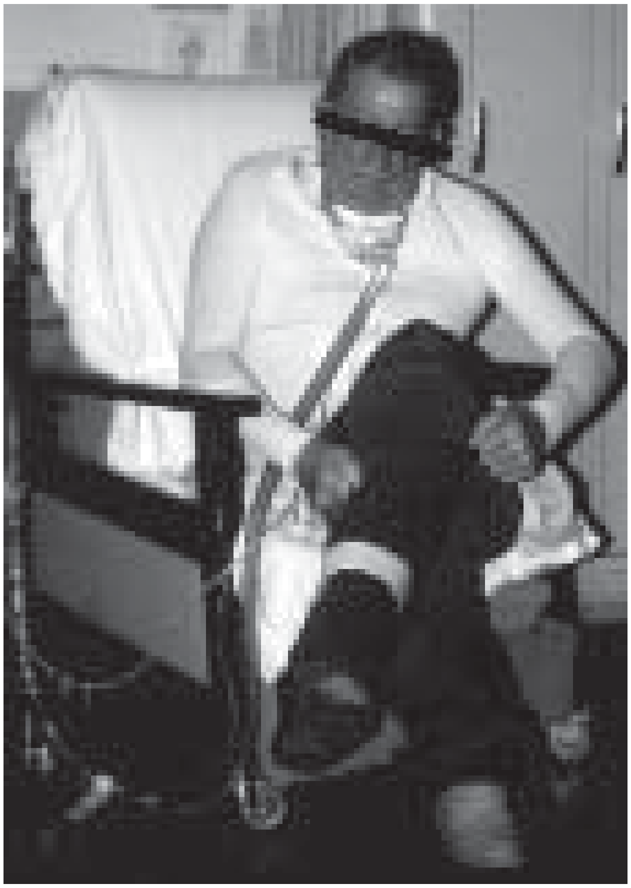

Fig. 10-106. The casualty is independently donning gym pants without adaptations. He is using Tubigrip external vascular supports.

member. It can also be performed by the casualty and family alone. A home evaluation, with an emphasis on independence and safety, can identify any functional or environmental limitations. The casualty has the opportunity to use actual mobility aids and adaptive equipment in his own home to determine the level of assistance required postdischarge.

There are a number of home accessibility aids to facilitate independence. A ramp to enter the house is very important if a casualty is wheelchair bound. Stair glides are often used within a house to allow movement from one floor to another. Various types of high rise furniture leg extenders (for chairs and couches) increase the ease of transferring independently. There are lamp and light switch extensions that are helpful if a casualty has decreased finger dexterity or is functioning from a wheelchair. Also, a variety of turning adaptations exist for the operation of faucets, stoves, and radiators when a weak grasp is present.

As the burn casualty continues in the rehabilitation process, he will generally resume kitchen activities (Figure 10-107). There are special devices that can be used for safety when operating the oven. There is a push/pull device that is used to manipulate hot oven racks when there is limited finger usage or sensory deficits. Long oven mitts made out of flame retardant fabric offer protection for sensitive skin. Kitchen roll carts are effective in moving hot or heavy pans and dishes. If the cart is sturdy it can be used like a wheeled walker for stability. If an individual is cooking from a wheelchair, a positioning mirror over the stove allows him to see the burner to cook safely.

There are a variety of devices for use in the kitchen if the casualty has only one functional hand. There are pan and bowl holders that provide stability to compensate for the inability to hold with a nondominant hand. Dycem, the nonskid surface, provides stability under plates and bowls. The adaptive cutting board has a nail to stabilize items for cutting and a built-up corner to hold bread while buttering. A rocker knife, with a sharp, rounded blade, is useful with the use of one hand. There are many types of special openers for jars, cans, or bottles that can be used when a casualty has only one functional hand or decreased hand strength or control.

As with many of the other functional activities, there are adaptive housekeeping aids that compensate for decreased ability to reach or grasp. Extended dustpans with brushes, long reach sponge mops, and extended dusters are available for independent housekeeping. There is also a housekeeping cuff with Velcro attachments that permit an individual with a weak grasp to hold a broom or mop handle.

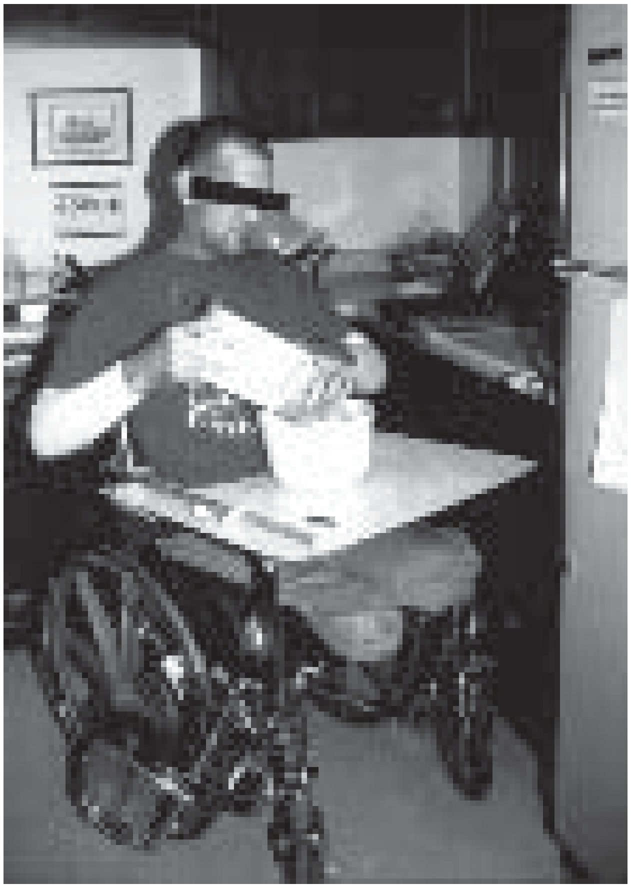

Fig. 10-107. The casualty is doing kitchen activities from his wheelchair. Note the use of a lapboard.

Communication

In the wound maturation phase of healing some of the earlier communication devices will not be necessary while others will continue to be required. At this stage, when the casualty is able to be out of bed in a seated position, typing and computer activities can be explored. These prevocational activities can be very important aspects of the rehabilitation process.

There are several adaptive aids that are useful with keyboard usage. Typing aids that fit over the hand and depress the keys are commercially available or can be custom made. They are useful when fine motor dexterity is lacking and the casualty is unable to access the keyboard in a traditional way.

Keyboards can be mounted on special devices if an individual has limited range of motion in the shoulders or elbows for accessing the keyboard. There are also detachable keyboards for when a person is unable to access the keys in the usual location. Key guards are special devices that are useful with individuals who have impaired coordination. The keyboard separates the keys and allows depression of the correct keys. A keylock is a device for one-handed users to simultaneously depress multiple keys. There are a variety of special switches to operate the environmental controls or activate the computer if an individual cannot directly operate the keyboard.

Functional Mobility

During the wound maturation phase of rehabilitation the casualty becomes more mobile as part of the exercise program. A transfer or gait belt is generally used around the individual's waist for stability during early phases of ambulation. Sometimes assistive devices for ambulation are used at this stage. For example, straight or quad canes or regular, tall, platform, or wheeled walkers. Frequently, these devices are discontinued as the casualty progresses to independent ambulation.

A casualty who is severely burned or has lower extremity amputations may use a wheelchair. Wheelchairs are available in a variety of models including standard, reclining, narrow, lightweight, and amputee models. In severe cases, electric wheelchairs can be used. For each type of wheelchair there are numerous options regarding additional supports, special seating systems, and accessories. Wheelchair supports can be designed specifically for an individual: headrests, lateral supports, adjustable height feet rests, and wheelchair safety straps. The correct seating system is extremely important for the burn casualty to protect the fragile skin and prevent decubiti. There are special cushions that can be placed over solid seats and back inserts. Cushions composed of gel, foam, or neoprene are helpful in maintaining skin integrity. There are several cushion manufacturers including JAY (Jay Medical), and ROHO (Roho Inc.). Wheelchair accessories include lapboards, arm troughs, sliding boards, and special gloves. Lapboards, which are available in many styles, including stand, clear, hinged, and swingaway, provide a desk top area for feeding or writing. Arm troughs, available in regular, foam elevating, and rotating models, are beneficial in maintaining appropriate upper extremity position and controlling edema.

Driving is a functional activity that should be performed cautiously. A physician's recommendation is made after psychoactive drugs, including analgesics, are discontinued. A driver's evaluation is beneficial in cases of severe burns to further determine the safety of the individual and the need for any adaptive equipment. An evaluation provided at an accredited facility can provide information regarding reaction time; the need for modifications in neck, face, and upper extremity orthoses; and resources and training with special equipment.

There are many types of adaptive equipment for driving. Hand controls for the accelerator and brake are used when a casualty has limited or no lower extremity function. Nonskid material can be placed on the wheel for a better gripping surface. Steering wheels with special rings and knobs, hooks or lever extensions for brakes, turn signals, gear selectors, and large side mirrors are available.

If a casualty is in a wheelchair, there are vans that can be driven independently with special controls. Portable ramps facilitate independence with getting in and out of the van. If fine motor dexterity is lacking, there are special car door openers and key turns that can be used to facilitate manipulation of these items.

Recreation

Recreation is an important functional activity. The benefits, in addition to decreased pain during exercise, include an appropriate physical or emotional outlet for anger, the enhancement of self-esteem, and increased feelings of accomplishment (see Figure 10-63). Card holders and card shufflers are available if someone has decreased grasp or one-handed usage.

In the wound maturation phase of healing, a burn casualty can begin to participate in organized games and sports or health clubs. This participation should be incorporated into a graded exercise program. The games and sports can have many physical goals such as increasing AROM, strength, coordination, sitting or standing tolerance, and endurance. Sports provide opportunities to improve attention, concentration, and problem solving abilities, as well as an opportunity for social interaction between casualties, families, other casualties, and friends.

Intermittent compression using elastic wraps or mechanical devices such as a Jobst Intermittent Compression Pump or a Wright Linear Pump (Wright Co.) can be used in the maturation phase as described in the acute phase. The mechanical devices should be considered when edema persists and simple compression and elevation does lead to resolution. These devices have been used successfully to attain skin closure of chronic ulcers secondary to chronic edema caused by lymphatic and venous insufficiency. In this phase, inflammation is probably less of a factor than in the acute phases. The underlying problem may well be more secondary to reduced lymphatic or venous flow secondary to thermal damage of underlying subcutaneous tissues or excision to fascia. Therefore, these pumps may be needed on a lifelong basis. If this is the case the casualty will need specific instruction in home use prior to discharge.

Functional electrical stimulation (FES) has been used in rehabilitation with the goals of retarding disuse muscle atrophy, reducing contractures due to weak muscles, and increasing venous and lymph flow in the stimulated muscles. In the burn casualty the goals would be similar. The skin must be healed enough to tolerate the electrodes and conductive gel or tape needed to hold the electrodes in place as well as to tolerate the current necessary for stimulation. Use of FES has been sporadic in rehabilitation in general as well as in burn rehabilitation. However, late improvement in objectively measured hand function was reported in one pilot study using FES in burn survivors with severe hand injuries.[143] This technique should be considered in this phase of burn care when the more common therapeutic exercise modalities are not providing the desired outcome.

Psychosocial Adjustment During the Wound Maturation Phase

Psychosocial issues continue throughout the wound maturation phase of healing. The soldier at home or in the rehabilitation center learns how to return safely to duty and routine life despite his injury. Physical changes are often bothersome, and physical fitness is now a prolonged, daily struggle; reconditioning may take place at a much slower pace than before the burn.[144]

Normal responses to a major burn injury often include crying, degrees of fear, depression,[87] grief, loss of hope, and other reactions unfamiliar to the soldier. Discharged patients report it takes about 6 to 7 months of being at home before they can cope, as they did before the injury, with emotions and activities that require concentration. Distractibility gradually subsides. Impatience, irritability, and frustration are common. The casualty can learn to accept gradually improving function instead of perfection in activities. Family members often can help sort out the realistic reactions from overreactions.

Psychological adjustment for returning to work in an area where the injury occurred must be considered. Few people will return easily to the injury site. Referral to a psychologist is appropriate. Return to duty at the earliest possible time maximizes the benefits of buddy support, as well as routine and work. Strength and coordination improve much sooner from work than from therapy. Ego strength and social interaction are also improved with return to duty. Feelings of anger, fear, loneliness, or helplessness bring pain acutely to the patient's awareness.[145] The soldier benefits from sharing personal experiences of pain relief. Former burned patients report helpful assistance from participating in a trauma survivors group. With leadership from social workers and psychologists or both, the group is able to deal positively with symptoms such as disturbing dreams, appetite disturbance, difficulty with sleep, feelings of estrangement or detachment, recurrent intrusive memories of the event, memory impairment, difficulty concentrating, reluctance to accept a changed body image, decreased interest in sex, sensitivity to loud noises or other cues related to the accident, irritability, and fear of returning to field duty or other work. Patients are often referred for group participation when these symptoms are noted by the casualty, family members, or involved staff members.

A minor burn affecting an exposed area of the body such as the face or neck may be more psychologically devastating than a severe burn that can be covered with clothing. In the event of disfigurement or loss of a body part, the casualty normally grieves these losses. Extensive burns produce one of the most devastating and dehumanizing injuries.[146] In addition, premorbid psychiatric disorders, alcoholism, or chemical abuse are observed more often in burned patients than in other populations.[147] Preburn psychiatric morbidity is associated with poor postburn psychosocial adjustment.[148] Burn severity does not usefully predict psychosocial outcome.[148]

A high incidence of PTSD among recently burned patients has been noted.[148] This syndrome is defined in the DSM III[149] with specific criteria. The burn patient responds to a recognizable stressor that evokes the distress symptoms. In addition, he often experiences vivid, intrusive dreams or recollections of the incident. Other frequently noted characteristics are an exaggerated startle response, impaired memory, concentration problems, avoidance of cues of the accident, and withdrawal from normal social interaction, chores at home, tasks at work, or participation in active duty. Treatment is aimed at giving the soldier as many choices as are reasonably possible during recovery, thereby relieving a sense of helplessness. Stress reduction strategies and goal directed individual counseling are also beneficial. Short term pharmacological intervention may also be appropriate. It is helpful for the soldier returning to active duty to know that exacerbation of PTSD may occur following such events as the dedication of the Vietnam War Memorial.[150] Reading about a similar burn injury may also revive PTSD symptoms.

Return to active duty improves the casualty's self-concept. The burn physician must consider the duty tasks and the progress of the wound when recommending return to active duty (Figure 10-108). Food handlers must remain off-duty until open areas are closed and wound cultures reveal no pathogens. Heavy laborers may need job modifications when they first return to duty. Often a transition period of half-day work progressing slowly to full-time work is needed. Rehabilitation counselors, nurses, or OTs can assist with changes in the work setting when these are needed. The adaptations are often inexpensive: for example, placing a foot stool near a bench or counter, on which the soldier places his alternate foot for half-hour periods; or arranging items within the reach of the soldier so he will not have to reach beyond his center of balance when he first returns to duty.

Appraising the extent of the burn injury and objectively estimating residuals that affect performance are most accurate when based on objective criteria[151] and experienced prediction. Rating permanent impairment is a physician's function. Disability or handicap is related to performance loss, preinjury age, education, economic and social situations, sex, and the burned person's attitude toward recovery. The physician has the final responsibility in determining when it is medically safe for the soldier to return to active duty.

Nutritional Care of Burn Patients

The burn injury results in major changes in metabolism that are believed to be largely hormone mediated due to increased catecholamines, glucocorticoids, and glucagon-to-insulin ratios. The metabolic alterations include increased gluconeogenesis, proteolysis, ureagenesis, and decreased lipolysis and ketone utilization. In addition, destruction of the skin barrier results in physiologic losses of heat, water, and water soluble nutrients. These changes result in increased energy expenditure, increased nitrogen losses, and changes in nutrient metabolism.

The hypermetabolic-catabolic response becomes evident within 2 to 3 days of the burn injury, reaches a peak between 4 and 20 days, and then gradually decreases with wound closure and healing. The magnitude and duration of the response can be decreased, though not totally ameliorated, by placing the patient in a warm environment, providing adequate pain relief, completing early wound closure, applying occlusive wound dressings, and preventing sepsis. Thus, nutritional support should be initiated early, meet estimated needs, and be monitored closely to promote healing.

Nutritional support for the burned patient may include any combination of diet, oral supplements, and tube or parenteral feeding, or both. Nutritional care includes early assessment of nutritional requirements, development of an appropriate nutritional care plan, and close monitoring of the adequacy of nutritional intake.[152]

Nutritional Assessment

Traditional indexes of nutritional status such as anthropometric measurements, weight, plasma proteins, and immunocompetence are of limited value when assessing burned patients since they are altered by the injury itself. Thus, nutritional assessment should be aimed at estimating current nutritional requirements.

Energy. Energy needs can be estimated using equations based on body size, age, and activity level or percentage of burn, or both; it can also be measured using indirect calorimetry. For children, energy needs for growth should be included in total energy estimates. Preburn weight should be used whenever possible because postburn weights are affected by edema and bulky dressings. Exhibit 10-10 summarizes the most commonly used equations for estimating energy requirements in adult and pediatric patients that were identified in a recent survey of US burn centers.[153-163]

A criticism of these equations is that they frequently overestimate energy needs, particularly for patients with greater than 50% TBSA. Studies[164,165] using indirect calorimetry to measure energy expenditure indicate that there appears to be an upper limit of energy expenditure at approximately twice basal energy estimates.

Serial measurements of resting energy expenditure by indirect calorimetry are increasingly being viewed as the best method to determine the energy requirements of a burn patient. Indirect calorimetry

PATIENT QUESTIONNAIRE

PHYSICAL ISSUES TO BE ADDRESSED BEFORE RETURN TO WORK OR ACTIVE DUTY

Yes	No		
			Epithelial system
____	____	1.	Is the skin intact?
____	____	2.	Are pathogens absent from the open areas (if the skin has open areas)? Which pathogens are present?
____	____	3.	Is the skin moist, supple and resistant to low humidity?
____	____	4.	Is the partial thickness healed skin durable enough for the person's occupation?
____	____	5.	Are the sheet and meshed grafts durable?
____	____	6.	To what extent can the employee's skin tolerate exposure to extremes of heat?
____	____	7.	To what extent can the employee's skin tolerate exposure to extremes of cold?
____	____	8.	Does the healed skin tolerate unfiltered ultraviolet light?
____	____		Has protective re-pigmentation developed?
____	____	9.	What is the tolerance to contact with chemicals or petroleum products?
____	____	10.	What is the skin's tolerance to exposure to vapors such as ammonia in cleaning solvents or animal urine
____	____	11.	Is the healed skin resistant to dust?
____	____	12.	Are scar control garments still needed? Do these interfere with work?
____	____	13.	Is the skin cosmesis altered?
____	____		Has redness faded?
____	____		Are hypo- or hyperpigmentation permanent?
____	____		In what areas?
____	____		Is the skin thinned, wrinkled?
____	____		Is the mesh graft pattern faded?
____	____		Has the healed donor changed color?
____	____	14.	Is unsightly disfigurement present, especially in the facial and hand areas? Can it be camouflaged?
____	____		Does it interfere with job performance?
			Cardiovascular/respiratory system
____	____	15.	Is chronic edema a problem in dependent tissues? Are external vascular supports needed?
____	____		Do they interfere with job performance?
____	____	16.	Is there loss of respiratory capacity?
____	____	17.	Has normal endurance returned?
____	____		Does tiredness interfere with job performance? How many hours of work can the employee tolerate?
			Musculoskeletal system
____	____	18.	Are joint contractures present? Do these interfere with job performance?
____	____	19.	Does the employee exhibit decreased eye-hand coordination and impaired dexterity?
____	____	20.	Has the patient achieved maximum benefits from therapy?
____	____		Is strength normal for age group? Does weakness interfere with job performance?
____	____	21.	Have accompanying bone fractures, osteomyelitis or septic joints healed?
____	____	22.	Are amputations present? Has the prosthesis been fitted? Is the patient using it in a coordinated, useful
____	____		way?
			Nervous system
____	____	23.	Does the employee have residual pain? Does it interfere with job performance? Prolonged activity?
____	____		What pain medications is the patient taking?
____	____	24.	Is the employee's sensation changed?
____	____		Has he learned to compensate for this?
____	____	25.	Is brain function changed?
____	____		Is the patient depressed?
____	____		What psychotropic medications is the patient taking?
____	____	26.	Is there fear of returning to work or places where similar cues as the place of injury are encountered?
____	____	27.	Is chronic itching resolved?
____	____		What itch medications is the patient taking?
____	____	28.	Is sexual dysfunction and changed body image being resolved?
			Eye, ear, nose, and throat systems
____	____	29.	Are there any changes in hearing? Do they interfere with job performance?
____	____	30.	Does the patient hear "buzzing," ringing or other sounds? Does it interfere with job performance?
____	____	31.	Is visual impairment present? Does it interfere with job performance?
____	____	32.	Does the patient have "dry" eyes? Does this interfere with vision or cause pain?
____	____	33.	Does the patient have frequent nose bleeds?
____	____	34.	Does the patient have a dry nose?
____	____	35.	Does the patient have hoarseness?
____	____		Adequate vocal volume?
____	____		Proper enunciation to be understood?

Fig 10-108. The patient questionaire used when the burn physician is evaluating the casualty for readiness to return to duty.

EXHIBIT 10-10

FORMULAS FOR ESTIMATING ENERGY REQUIREMENTS

ADULTS
    Modified Harris-Benedict Equations (BEE)*
        Long: BEE x activity factor x injury factor[1]
            Activity factor = 1.2 in bed; 1.3 out of bed
            Injury factor = 2.1 severe injury (> 45% TBSA)
        Wilmore: BEE x Injury factor for 20% to 45% TBSA = 1.6 to 2.0[2]
        Bell: BEE x 2[3]
    Curreri Formulas
        Ages 16 - 59 years: 25 kcal/kg + (40 x % TBSA)[4]
        > 60 years: 20 kcal/kg + (65 kcal/kg x % TBSA)[5]
CHILDREN
    Curreri Junior Formulas[6]
        0 to 1 year = Basal Calories + 15 kcal x % TBSA
        1 to 3 years = Basal Calories + 25 kcal x % TBSA
        3 to 15 years = Basal Calories + 40 kcal x % TBSA
    Galveston Formulas (Hildreth)
        Infants < 1 year: 2,100 kcal/m^2 + 1,000 kcal/m^2 burn[7]
        2 to 12 years: 1,800 kcal/m^2 + 1,300 kcal/m^2 burn[8]
        Teens: 1,500 kcal/m^2 + 1,500 kcal/m^2 burn[9]
    Wolfe Formula[10]
        BEE x 1.55 to 2

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*Harris-Benedict Equation (Basal Energy Expenditure, BEE)
    Male: 66.47 + [13.75 x (Wt in kg)] + [5.00 x (Ht in cm)] – [6.76 x (Age in years)]
    Female: 655.10 + [9.56 x (Wt in kg)] + [1.85 x (Ht in cm)] – [4.68 x (Age in years)

Sources: (1) Long CL, Schaffel BS, Geiger JW, Schiller WR, Blakemore WS. Metabolic response to injury and illness: estimation of energy and protein needs from indirect calorimetry and nitrogen balance. *J Parenteral Enteral Nutrition*. 1979;3:452-456. (2) Wilmore DW. *The Metabolic Management of the Critically Ill*. New York: Plenum Medical Book Co; 1977. (3) Bell SJ, Wyatt J. Nutritional guidelines for burned patients. *J Am Dietetic Assoc*. 1986;86:647-653. (4) Curreri FW, Richmond D, Marvin J, Baxter CR. Dietary requirements of patients with major burns. *J Am Dietetic Assoc*. 1974;65:415-417. (5) Adams MR, Kelley CH, Luterman A, Curreri PW. Nutritional requirements of the burned senior citizen: The Curreri senior formula. *Proc Am Burn Assoc*. 1987;19:83. (6) Day T, Dean P, Adams MC, Luterman A, Ramenofsky ML, Curreri PW. Nutritional requirements of the burned child: The Curreri junior formula. *Proc Am Burn Assoc*. 1986;18:86. (7) Hildreth MA, Herndon DN, Desai MH, Broemeling LD. Caloric requirements of burn patients under one year of age. *Proc Am Burn Assoc*. 1992;24:153. (8) Hildreth MA, Herndon DN, Desai MH, Broemeling LD. Current treatment reduces calories required to maintain weight in pediatric patients with burns. *J Burn Care Rehabil*. 1990;11:405-409. (9) Hildreth MA, Desai MH, Herndon DN, Duke MA. Caloric needs of adolescent patients with burns. *J Burn Care Rehabil*. 1989;100:523-526. (10) Gorman M, Broemeling L, Herndon DN, Peters EJ, Wolfe RR. Estimating energy requirements in burned children: A new approach derived from measurements of resting energy expenditure. *Am J Clin Nutrition*. 1991;54:35-40.

involves the use of a portable metabolic cart that measures respiratory gas exchange from which resting energy expenditure is calculated. This measured energy expenditure includes the increased needs due to the injury but not the energy needed for activity because it is performed when the patient is at rest. Additions of 10% to 30% for activity are recommended to estimate total calorie needs. A respiratory quotient (RQ) also can be derived that allows estimation of overfeeding ($RQ > 1.0$) or underfeeding ($RQ \sim 0.7$). Indirect calorimetry is, however, relatively expensive, time consuming, and requires an experienced technician to obtain reproducible results.

Whatever method for estimating energy need is used, it should be reevaluated at least weekly during the early recovery stages. Overfeeding can result in increased respiratory requirements and carbon dioxide production, hyperglycemia, osmotic diarrhea, and fatty liver. Underfeeding can affect wound healing and immunocompetence. Several studies have measured energy needs over time following the burn.[165]

Protein. The burn injury results in profound changes in protein metabolism with an increase in liver synthesis of acute phase proteins. Energy needs are met by muscle proteolysis, which results in muscle wasting. In addition, significant amounts of protein are lost through the open wound. Although optimal protein requirements are unknown, current protein recommendations for adults and children are 20% to 25% of total calories or a calorie to nitrogen ratio of 100:1 to 150:1.[166-168] This is approximately 80% to 100% above maintenance needs. However, it remains unclear if increasing protein intake results in increased anabolism.[169] Protein intake should be adjusted serially, based on nitrogen balance, whenever possible.

Recent studies have focused on the need for and role of specific amino acids in nutritional support of the burn patient. Arginine supplementation has been shown to improve cell mediated immunity and wound healing and decrease morbidity and mortality in several studies.[170-172] Arginine at 2% of calories is recommended by those researchers.[172,173] Glutamine supplementation is advocated by others because of its role in preserving gut integrity, and decreasing translocation and wound infections.[174] Some researchers advocate 0 to 40 g per day.[175] Use of high branched-chain amino acid formulas have not been found to be beneficial to burn patients.[176]

Carbohydrate and fat. Alterations in carbohydrate metabolism during the acute postburn phase include increased glucose production from gluconeogenesis. Carbohydrate is the primary energy substrate but should be limited to approximately 5 to 7 mg/kg/min, the maximum oxidation rate in adults.[177] At higher rates, hyperglycemia and osmotic diuresis can occur, and the carbohydrate is converted to fat and the sequelae associated with overfeeding.

The optimal amount and type of lipid to use in nutritional support of the burn patient is controversial and currently the subject of extensive research. During the acute postburn phase, there is a decrease in lipolysis since protein is the preferred fuel source, and an increase in serum free fatty acids and triglycerides. Lipids are a concentrated source of calories for the burn patient, but high levels of lipid intake, especially long chain polyunsaturated fats, may impair immune function. Modification of lipid intake with omega-3 fatty acids have been shown to improve immune competence and tube feeding tolerance.[178] Medium chain triglycerides may be helpful in decreasing omega-6 fatty acid intake, yet provide an easily absorbable source of fat calories. Many enteral formulas contain medium chain triglycerides as part of their fat content. Current recommendations are to limit lipids to about 15% to 20% of total nonprotein calories.[166,178,179]

Vitamin and mineral requirements. Specific requirements for vitamin and minerals have not been established although it is thought there are increased needs for at least those nutrients involved in wound healing and tissue synthesis (vitamins C and A, and zinc). Provision of a vitamin-mineral supplement equal to the recommended dietary allowances (RDA) is commonly recommended. Additional daily supplements of one gram of ascorbic acid, 10,000 IU of vitamin A, and 250 mg of zinc sulfate are used at many burn centers for adults.[153,180,181] Recommendations for pediatric patients include a daily multivitamin equal to the RDA for age, vitamin C at 5- to 10-fold the RDA, and 2-fold the RDA for zinc.[182]

Nutrition Care Planning

Development of a nutritional care plan involves selection of the appropriate route of nutritional support and selection of the specific formula, diet, or supplement to be used. Early enteral support within the first 24 hours is the preferred method of nutrition support as it may attenuate the hypermetabolic response.[183]

Oral high calorie and high protein. Patients with small percentage burns of 1% to 20% TBSA have modest increases in calorie and protein needs that can usually be met by diet alone or with the addition of between-meal supplements. The care plan should include individualization of meal plans and meal times to facilitate maximum intake because routine burn cares and rehabilitation frequently interfere with scheduled hospital meals. Calorie counts and documentation of supplements actually consumed are important for monitoring the adequacy of intake.

Enteral nutrition support. Adult patients who have burns greater than 20% to 30% TBSA, will usually require nutritional support in addition to diet. Pediatric patients with burns greater than about 10% TBSA will also likely need nutritional support. Children, especially young children, with burns of an even relatively small percentage TBSA may refuse to eat and thus require tube feeding. The enteral route is preferred for reasons of safety, better utilization of nutrients, preservation of gut integrity, and lower cost. Recent studies[183,184] of initiation of feeding within 6 to 12 hours of injury indicate the desirability and safety of early enteral feeding. Feeding protocols that outline formula selection, initial goals, and initiation rates are helpful in starting enteral nutrition support early.

Selection of the appropriate route of enteral nutrition (nasoenteric, nasogastric, gastrostomy, or jejunostomy) depends on the aspiration risk, patient's condition, and the expected duration of enteral support. Nasoenteric feedings are preferred by many centers since they afford decreased risk of aspiration and can be continued during times of gastric ileus and surgical procedures. Small bore feeding tubes should be placed under fluoroscopy past the ligament of Treitz. Tubes made of erythrothane or polyurethene are suitable for long term use since they remain soft and pliable. Gastrostomy and jejunostomy routes are not used frequently in the acute phase because of the increased risk of wound infections in burn patients.

A moderately low fat, high protein formula is recommended. Elemental or hydrolyzed protein formulas are usually not necessary because digestion and absorption are usually normal in burn patients. Isotonic formulas can be initiated full strength at low rates (25 to 50 cm^3/h) and advanced to the goal over 24 to 48 hours. Complications include aspiration, diarrhea, metabolic abnormalities, and mechanical problems. Usually these can be treated or reduced without discontinuing the tube feeding.

Parenteral nutrition support. Parenteral nutrition support should be reserved for patients who have a nonfunctioning gastrointestinal tract or as a supplement to enteral nutrition when requirements cannot be achieved enterally. Close attention to line care is essential since the burn patient has a high risk of developing infection and sepsis.

Nutritional Monitoring

Monitoring the adequacy of nutritional intake is an essential component of the nutritional care of the burn patient regardless of the route of support. Because no single parameter accurately indicates nutritional status and most parameters are altered by the burn injury, several indexes should be selected for monitoring. These parameters are followed serially for trends rather than using their absolute values. Weight, nitrogen balance, calorie counts, and visceral protein status are useful when evaluated globally, along with consideration of the patient's clinical situation. Weight changes should be evaluated in relation to preburn weight and as a trend. Nitrogen balance requires an accurate 24-hour urine collection and a record of the patient's protein intake. Nitrogen excretion should be modified to include open wound losses. Two formulas proposed by Bell and Waxman are shown in Exhibit 10-11.[185,186]

Calorie counts are particularly important when a patient is being transitioned from parenteral or enteral support to oral diet or when diet is providing all of the nutritional support for the patient with significant surface areas burns.

EXHIBIT 10-11

NITROGEN BALANCE EQUATIONS

Nitrogen balance = Nitrogen intake - (total urinary Nitrogen [TUN] + fecal nitrogen loss + wound nitrogen loss)
(If TUN is not available, use urinary urea nitrogen [UUN] plus 1 to 2 g for nonurea nitrogen)

Equations to estimate wound nitrogen Loss:

Bell[1]:
< 10% burn = 0.02 gm nitrogen/kg/day
11% to 30% burn = 0.05 gm nitrogen/kg/day
> 31% burn = 0.12 gm nitrogen/kg/day

Waxman[2]:
Postburn days 1 to 3 = 0.3 x BSA x % burn
Postburn days 4 to 16 = 0.1 x BSA x % burn

BSA: body surface area in m^2

Sources: (1) Bell SJ, Molnar JA, Krasker WS, Burke JF. Prediction of total urinary nitrogen from urea nitrogen for burned patients. *J Am Dietetic Assoc.* 1985;85:1100-1104. (2) Waxman K, Rebello R, Pinderski L, et al. Protein loss across burn wounds. *J Trauma.* 1987;27:136-139.

Visceral protein status is difficult to evaluate because all of the indexes (albumin, transferrin, prealbumin, and retinol binding protein) are abnormally low in patients with major burns. Albumin is the poorest indicator due to its long half-life (21 d) and susceptibility to fluid shifts. Transferrin (half-life ~ 8 days) or prealbumin (half-life ~ 2 days) are better indicators of the patient's response to nutritional intake. Trends should be evaluated along with other parameters. Nutritional support requires the cooperation and support of the entire burn team to plan, provide, and monitor adequate nutritional intake.

Neurologic Problems in Burn Patients

Neurologic problems, many of which are preventable in burn patients, are the result of many etiologies. It is important to understand the etiologies of these deficits so precautions can be taken for prevention. Early recognition and treatment may help to prevent permanent deformity. Peripheral neuropathies are frequent and, yet, the diagnosis is often missed and the clinical weakness or atrophy noted is attributed to disuse weakness as a result of prolonged hospitalization with periods of forced immobility. The incidence of peripheral neuropathy has been reported at almost 30%. The most frequently diagnosed neuromuscular abnormality is generalized peripheral neuropathy, which commonly presents as distal weakness in the upper and lower extremities. The patient's complaint, however, is usually lack of endurance and easy fatigability, not weakness. When weakness is noted, it occurs in the burned as well as unburned extremity. Clinically detected sensory deficits are uncommon. However, with electrodiagnostic testing, slowing of sensory nerve conduction velocities is detected. There is a greater incidence of peripheral neuropathies as the burn size increases above 20% TBSA in adults and 30% TBSA in children. Electrical burn patients develop neuropathies with smaller burns. The cause of generalized neuropathies is not entirely understood but is probably multifactorial, related to toxic, nutritional, and metabolic factors. Additional research is needed to discover preventive interventions for the generalized neuropathies. Localized neuropathies are of great concern because they are probably related to preventable causes such as compression or stretch injuries of a peripheral nerve or damage from intramuscular injections.[187]

Predisposing factors make some patients more susceptible to neuropathies: the aged are more prone to develop compression neuropathies because their peripheral nerves do not tolerate pressure well, and older patients are less mobile. Alcoholics and diabetics are also prone to neuropathies because of already diseased nerves.[188]

The most commonly affected peripheral nerves are the peroneal, ulnar, and brachial plexus. The peroneal nerve is prone to both pressure and stretch injury. It courses around the fibular head and is covered only by skin and superficial fascia. Prolonged sidelying in bed creates excess pressure along the course of the nerve. Additionally, this nerve can be compressed by orthoses or elastic compression applied too tightly over the fibular head. Bulky wraps used for immobilization postgrafting must be applied so as to limit compression and actually provide pressure relief to this area. Stretch neuropathies occur by improper positioning with the hips flexed, abducted, externally rotated with knee flexion, plantar flexion, and inversion of the foot. This "frogleg" positioning is assumed by the patient because it is the position of comfort for the burn casualty. Therefore, preventive measures include low air loss beds, rotating the sidelying bed position, and positioning the knees in extension and the feet in dorsiflexion when the casualty is supine. Hip trochanter rolls are used to position the leg, rather than knee rolls.

Ulnar neuropathies occur in a similar manner. The ulnar nerve is at risk of compression as it passes through the cubital tunnel at the elbow. When the elbow is pronated, the tunnel is narrowed. The burn victim commonly lies with the elbow elevated on pillows or arm troughs flexed at approximately 90° and pronated. The ulnar nerve thereby receives both external and internal compression. The subsequent damage to the ulnar nerve results in weakness of the ulnar intrinsic muscle of the hand, which causes a claw hand deformity with loss of sensation to the ulnar side of the hand. Prevention with proper positioning would include limiting the external pressure applied over the cubital tunnel as well as avoidance of the static pronated position.

Brachial plexus neuropathies are sometimes blamed on the use of strenuous stretching techniques. However, the problem is more likely caused by improper positioning. When the arm is flexed to 90° and externally rotated, the clavicle comes into close proximity to the first rib and can impinge on the plexus. This impingement can be prevented, however, by horizontally adducting (forward flexion) the arm to 30°. The staff should monitor a casualty carefully when this position is necessary for a prolonged period postgrafting. The arm may be safely positioned in abduction of up to 120° as long as sufficient horizontal adduction is allowed. The plexus is prone to injury in the operating room especially if the patient is supine and the arm is allowed to be abducted and not elevated above the level of the operating table. Also, if an axillary contracture is released, great care must be taken not to overstretch the also contracted neurovascular bundle.

Tourniquet injuries are seldom seen with the advent of pneumatic tourniquets in the operating room. However, these tourniquets, which are so important in providing a bloodless field necessary for tangential incision and grafting, must be used with care so as not to cause either direct pressure damage to underlying structures or cause ischemic injuries to distal tissues.

Bone and joint changes are common complications following thermal and electrical injuries.[189] Commonly noted changes include internal changes in bone, such as osteoporosis; bone necrosis; bone growth disorders in children; and periosteal bone formation. Early mobilization and weight bearing is thought to diminish the risk and severity of osteoporosis. Periarticular changes include heterotopic ossification and calcific tendonitis. Heterotopic ossification has been reported with an incidence between 13% and 23%. No definite etiology is known, although superimposed trauma or repeated minor trauma with local hemorrhage have been considered. Aggressive stretching with local trauma has also been implicated. Generally, periarticular ossification is noted in the area of deep burn, although it has been noted in areas distant from the burn; the elbow is the most common site, although the shoulder and hip are also common sites. It should be considered if there is a sudden onset of joint pain; swelling or redness may not be easily detected in the burn patient. When this diagnosis is made by plain radiograph or bone scan, the rehabilitation therapy consists of AROM exercises without stretching and orthotic positioning in the position of maximal function.

Joint changes such as septic arthritis and ankylosis occur when the injury occurs deep into the joint or when bacteremia seeds the joint. When a joint is thought to be infected, it should be positioned and rested using a static orthotic device in a functional position. If ankylosis occurs secondary to the septic process, the joint will then be at a maximal position of function. Dislocation occurs from improper positioning of a injured joint or, more commonly, by skin contracture. If a joint is subluxed by a skin contracture, orthotic management should be instituted immediately. If the orthosis cannot adequately control the deformity, internal fixation or release of the contracture or both should be strongly considered.

Plastic Surgery Strategies and Rehabilitative Considerations

Timing of reconstructive surgery and subsequent rehabilitation team efforts are a significant consideration during the rehabilitative phase of burn recovery. Correctly timed, the patient improves functionally, cosmetically, and psychologically. Incorrectly timed, the patient loses function, wastes valuable donor areas, and receives no benefit from the procedure.

Plastic surgeons avoid the words cosmetic and cosmesis, which continually emerge in the discussion of burn injuries. These descriptive words have connotations in American language that result in misinterpretation of the procedures being considered. Once a surgical procedure is termed “cosmetic,” it carries a tainted, frivolous identity. Almost no procedure for reconstructing the burn patient fits this category. Plastic surgeons fully understand the medical necessity of functional reconstructive surgery, which is vital for patients, to enable them to perform skillful work, recreation, and family life with self-confidence. This reconstruction for function should not be confused with “cosmetic” interventions.

In addition to reconstructive surgery, plastic surgeons recommend camouflage make-up. Cosmetic products that are used to enhance appearance and self-confidence for both male and female patients should not be considered unnecessary or frivolous.

The phases of recovery addressed by the plastic surgeon include the acute phase, during which the wounds are closing, and the wound maturation phase, during which the scars are maturing. The ideal time to undertake reconstructive surgery is after the scars have become mature. However, there are a few specific situations in which reconstruction must begin earlier. In many cases the patient wants reconstruction at an early date, and one must give a thorough explanation of the disadvantages of increased inflammatory scar deposition during the early scar maturation phase. As time progresses and the scars mature, the patient often becomes more satisfied with the appearance of the scars.[190] In addition, as the person becomes involved in former activities, he is less interested in prolonged interruptions for operations or in-hospital care.

The reconstructive surgeon and the patient select the most troubling functional deficits or disfiguring scars or both and discuss the possible correction. The patient takes an active part in the planning process. Surgical teams frequently perform multiple operations under the same anesthetic so that the time is used efficiently and recovery time is minimal. For instance, a 5th finger flexion contracture is released at the same time as a web space contracture is corrected with a Z-plasty on the same hand. Only one hand is operated on at a single sitting and early motion cases are not mixed with procedures that require immobilization. In most cases, external vascular support garments are not worn and compression is usually not helpful after the reconstructive operations. If difficulty with hypertrophic scarring develops after reconstruction, the team then starts using pressure, gel sheets, or intralesional steroids. It is important in discussing the rehabilitation of burn patients to understand the choices available for reconstruction. Generally the reconstructive surgeon determines the problem to correct, and then considers multiple ways of performing the corrections. The surgeon then chooses the optimal method for a particular patient as well as a backup procedure in case of complications or tissue loss postoperatively.

Skin Graft

Skin grafts for reconstruction are the most common source of covering tissue. For the best outcome, new donor sites should be available in unscarred areas, and these areas must be acceptable to the patient because of the resulting donor site scars. Thicker skin grafts are used for reconstruction than those in the initial skin grafts. The thin grafts contract far more than the thicker grafts but take with greater certainty. Any skin graft done when the scar tissue is active contracts severely, limiting the possibilities available to the plastic surgeon. The contraction in immature scars is related to the presence of the myofibroblast in the healing scar in contrast to the mature scar. In some areas of the body, contraction of the graft provides a poor functional outcome so that full thickness grafts are desirable. Such areas are the lower eyelids, dorsum of the fingers, nasal tip, ala, and upper lip. Reconstruction of the ala will require using composite grafts that include skin, fat, and cartilage.

Flaps

Skin flaps are frequently used in burn reconstruction when vital structures need coverage. These may be used in any phase of the reconstruction, and various flaps are used for different purposes. Musculocutaneous flaps or muscle flaps are often used acutely to cover bone, vascular grafts, or vital organs exposed by the burn itself. Musculocutaneous flaps are also used during the reconstructive procedures. Muscle provides excellent blood supply, new lymphatics, and thick composite coverage. In some cases the initial reconstructions are bulky and do not shrink adequately, so that the volume must be reduced at a later procedure.

Free flaps are used to provide blood supply to large avascular areas such as the scalp following electrical injury. These flaps require a microvascular anastomosis and a very specialized and individualized donor site. For example, a free flap of omentum has been used to cover a complete scalp defect. An overlying skin graft is then required (see Figure 10-20). Abdominal burns or gastrointestinal pathology may make this choice unwise; if so, a latissimus dorsi flap may be selected.[191,192] A thin, free flap including skin, such as a dorsalis pedis flap, may be better than a muscle flap reconstruction, which would require an additional overlying skin graft. Free flaps are useful in all phases of burn healing and allow for a great deal of creativity and flexibility for the plastic surgeon.

Axial flaps are long, cutaneous flaps that have an anatomically recognized artery and vein within the flap itself. The flap is either turned, rotated, or moved into position. Axial flaps are usually used for hand procedures such as a pollicization or island pedicle finger pulp reconstruction.

Random or local flaps were the earliest flaps used, do not have a recognized artery, and are used almost anywhere on the body surface. The skin flap survives on the subdermal plexus of vessels. These flaps may undergo a delay procedure to enlarge the flap. Tissue expansion may be utilized to expand the size of tissue available.

The Z-plasty is a procedure using multiple small flaps to lengthen a contracture. Z-plasties may have multiple flaps set around a specific joint or have multiple flaps set along a contraction line. Occasionally, these flaps are mixed with small skin grafts.

Timing of reconstructions may depend on the type of burn wounds. Chemical and thermal burns may need no reconstruction at all as they slowly improve. Radiation burns, in contrast, tend to be chronic and gradually worsen. These injuries require late debridement and flap coverage years after the initial trauma. Skin grafts in these cases are ineffective; musculocutaneous flap coverage to provide a new blood supply is ideal.

Electrical burns often require reconstruction during the acute phase of burn injury. These burns ex-

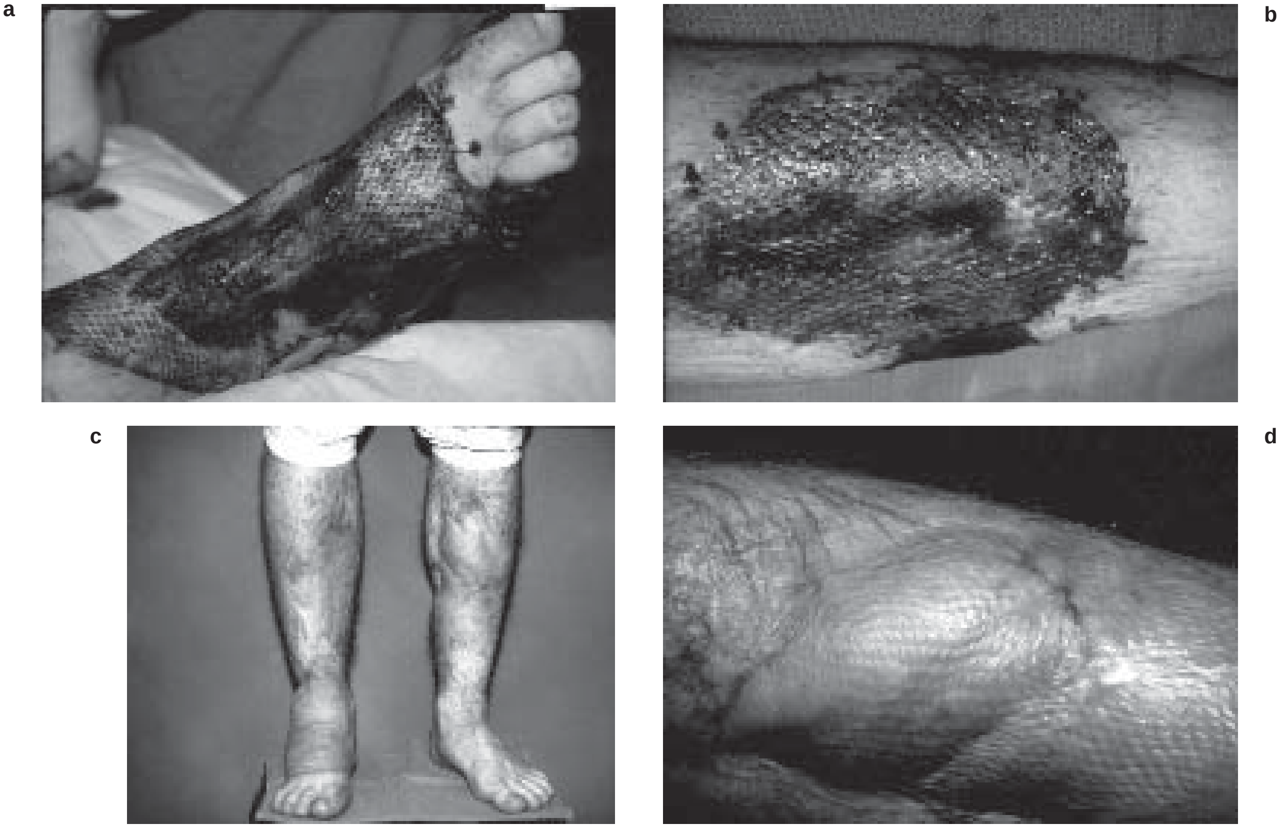

Fig. 10-109. Care of deep thermal burn of the leg. (**a**) Deep thermal burn of right foot dorsum with tendon involvement. (**b**) Exposed tibia on the same patient's opposite extremity. (**c**) Closure of both wounds 10 months later with free flap to foot. (**d**) Closure of open tibia with gastrocnemius muscle flap.

pose vital structures such as tendons, bones, or viscera, which must be covered. Rarely will split thickness skin grafts provide the quality of coverage needed, and flaps are vital to introduce new blood supply during the first few weeks. Musculocutaneous flaps and free flaps are the most adaptable methods for coverage of electrical wounds (Figure 10-109).

Release of an axillary contracture is related to function of the shoulder. Resurfacing of the forehead is related to the patient's appearance. In the face, reconstruction and function begin to merge. The function of the facial skin is to identify the individual, transmit emotion in communication, protect corneas, and form the mouth and nose. Eyelids, vital to the protection of the eye, may require reconstruction at a very early stage in the acute phase of burn recovery. To correct eyelid eversion or contraction, full thickness donor skin must be obtained in sufficient quantity to replace the eyelid skin. If hair-bearing skin or scarred skin is used, the results may give an unacceptable appearance and rapid reoccurrence of ectropion. Reconstruction of aesthetic units of the face during the acute phase gives an optimal appearance that may not be matched by any other procedure again until the patient has completed the maturation phase of burn wound healing. In the interim, facial orthoses, for example, transparent face masks, are the primary option for improving the appearance.[193]

Timing of functional hand burn surgery is fairly independent of reconstructional considerations for appearance. In society, hands are necessary for their function and not for their appearance in most situations. After initial closure of the wound, motion of fingers is vital, and surgery assumes a secondary role until the wound has become mature. Once the skin begins to feel supple, reconstruction can be undertaken. Skin grafts to the hand need immobilization for 10 to 14 days, followed by aggressive remobilization utilizing prolonged stretch and AAROM, AROM, and CPM equipment as necessary.

Extensor tenolysis is undertaken only when skin coverage is good. The patient must start range-of-motion exercises within 24 to 48 hours of extensor tenolysis surgery. Web space reconstruction using Z-plasties is managed like skin grafts with 10 to 14 days of immobilization and 6 to 8 weeks of spacers at night. It is not unusual to need an unexpected skin graft during a web space release because, as the scars are incised and defects are opened, the need for additional skin coverage becomes obvious. The patient's needs influence the timing of reconstruction, and all problems cannot be addressed at one sitting. Functional problems with hands, eyelids, mouth, axillae, elbows, and neck are the most important to the well being of the patient. Muscles, tendons, and nerves shorten when the skin over a joint is contracted. In these cases, reconstruction takes a precedence even if the maturation phase has not been completed.

Facial features themselves are not reconstructed until the scar tissue has become inactive, supple, and mature. An example would be an electrical burn involving the commissura of the mouth, such as that which occurs from a toddler biting into an electrical cord or sucking on an outlet. These injuries result in quite extensive tissue destruction. Only after these open wounds are allowed to heal are any reconstructions undertaken. In many cases the spontaneous healing, with the use of a microstomia appliance, produces such an excellent result that no further reconstruction is necessary.

The rehabilitation personnel need to understand the objectives and timing of plastic surgical interventions in the burn casualty. They need to consult with the plastic surgeon and provide objective measures of function. Outcomes are improved when there is appropriate timing of the needed procedure. Proper postoperative rehabilitation care is best directed by the plastic surgeon who knows the surgical intervention and proper timing to resume therapy, splinting if needed, and independent activity by the patient.

CONCLUSION

This chapter was written to summarize the critical elements of burn rehabilitation of the injured soldier, and discuss and illustrate proper rehabilitation of the burned individual. It was written by a team of specialists including physicians, nurses, dieticians, physical therapists, occupational therapists, and psychologists. This gives it both a comprehensive approach as well as a flavor of team work which is needed for the successful rehabilitation in a complex patient.

It provides medical knowledge at the level of all team members so as to provide an overview for all caregivers.

It is hoped that it will provide an excellent field guide to all military personnel as well as a more comprehensive guide for personnel located in a designated burn treatment area. More information can be found by utilizing the references provided.

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Chapter 11

ORTHOTICS FOR THE WOUNDED COMBATANT

JUSTUS F. LEHMANN, M.D.*

INTRODUCTION

UPPER EXTREMITY ORTHOSES
  Hand Orthoses
  Orthoses for Reduction of Contractures
  Interphalangeal Joint Stabilizer
  Wrist Orthoses
  Devices to Provide Reach
  Static Orthoses in Support of Shoulder and Elbow
  Functional Orthoses for Shoulder and Elbow
  Basic Support of Shoulder and Elbow Orthoses

LOWER EXTREMITY ORTHOSES FOR NEUROMUSCULAR CONDITIONS
  Ankle-Foot Orthoses
  Knee-Ankle-Foot Orthoses
  Orthoses for Skeletal Insufficiency

CONCLUSION

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INTRODUCTION

The science of orthotics deals with the design and use of orthotic devices, that is, orthoses. Orthoses provide joint immobilization, augment weakened muscles, assist in normal joint biomechanics, and can improve gait and the ability to conduct activities of daily living (ADL). This chapter reviews upper and lower extremity orthoses that may be prescribed to treat war-injured military personnel.

Orthoses for upper extremities include those that assist the veteran who suffers from paralysis or paresis resulting from peripheral nerve injuries; from upper motor neuron lesions, such as traumatic brain injury (TBI); and spinal cord injury (SCI). Specific discussions will address orthoses commonly used by hemiparetic patients whose conditions result from TBI or stroke and orthoses commonly used by quadriplegic patients from high level SCI. The designs of upper extremity orthoses offer great flexibility, especially for the hand. Standard designs of hand orthoses can be modified and customized to provide for the need of the individual patient.

The use of lower extremity orthoses as treatment for neuromuscular conditions will be similarly discussed, beginning with orthoses for paralysis or paresis resulting from peripheral nerve or upper motor neuron lesions (such as in TBI or SCI) or any combination of these. The biomechanical function of the orthosis will be clearly related to specific gait problems. Factors such as appropriate adjustment of the orthosis, reduction of energy consumption, and functional improvements will be addressed. This discussion will include ankle-foot orthoses (AFOs), knee-ankle-foot orthoses (KAFOs), and hip stabilization orthoses. In addition, lower extremity orthoses designed for the alignment of injured joints or bones will be discussed in terms of maintaining alignment and limiting weight bearing.

To manufacture properly fitting and functioning orthoses, technical skill is required and a laboratory must be available. Therefore, only a limited number of the devices described can be used close to the war theater. Most of them should be used in treatment programs that primarily deal with recovery from the injuries.

UPPER EXTREMITY ORTHOSES

Conditions for which orthoses are commonly prescribed include paralysis or paresis caused by (1) peripheral nerve injuries; (2) upper motor neuron lesions, such as TBI; and (3) high level SCIs. In some cases, an orthosis may be used for any of the described applications. Therefore, orthoses for all three conditions[1-5] will be discussed here. For an upper extremity orthosis to be useful, two major functions must be provided: (1) grasp (and release) and (2) reach.

Hand Orthoses

Hand orthoses are primarily designed to provide grasp and release. The following six grasp functions of the hand are used frequently in daily activities: (1) fingertip prehension or palmar prehension; (2) lateral pinch; (3) 3-jawed chuck; (4) large grasp, spherical object; (5) large grasp, cylindrical object; and (6) hook grasp, as for carrying a suitcase (Figure 11-1).

Basic Principles of Attaching Orthoses to the Hand

Due to the greater mobility of a hand and upper extremity in comparison to a lower extremity, a major problem is the firm attachment of the orthosis so it can be used for functional purposes, as shown in Figure 11-2. The basic orthosis is positioned diagonally across the palm of the hand to accommodate a firm grip. It embraces the area of the fifth metacarpal and passes over the dorsum of the hand just beyond the third metacarpal. The third metacarpal is the most firmly anchored component of the palm of the hand, whereas the fourth and fifth are highly mobile in the palmar dorsal direction. To keep the orthosis from sliding off toward the ulnar side, the orthosis is anchored to the side of the second metacarpal; this is called a radial extension (see Figure 11-2). This type of orthosis, without an extension across the wrist joint, is called the basic opponens orthosis. It is held by a strap extending from the dorsal portion of the brace across the wrist proximal to the heel of the hand (Figure 11-3). If the orthosis extends across the wrist into the forearm, it is called a long opponens orthosis. Modification of these orthoses will substitute for many of the grasp functions of the hand.

Orthoses can substitute for grasp by (*a*) stabilization of unstable joints where motion is not needed, (*b*) transfer of power from available muscles

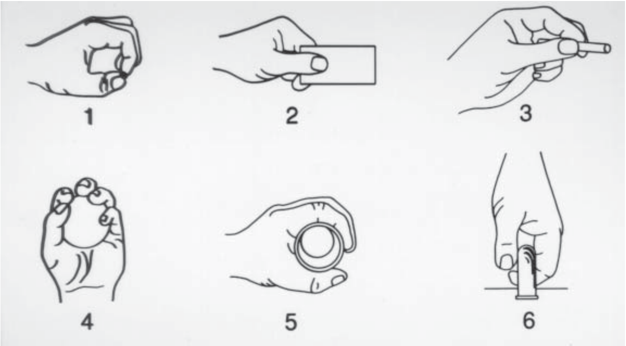


Fig. 11-1. Hand prehension patterns: (1) fingertip or palmar prehension; (2) lateral prehension; (3) 3-jawed chuck; (4) large object, spherical grasp; (5) large object, cylindrical grasp; (6) hook or snap. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965: slide 32: Various commonly used types of grasp.

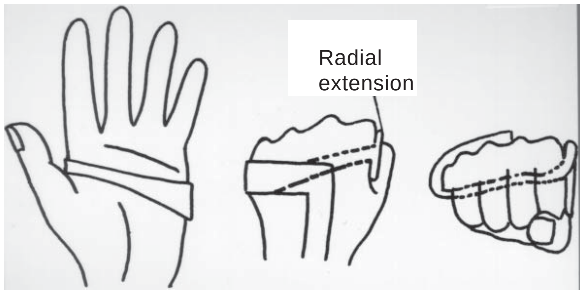
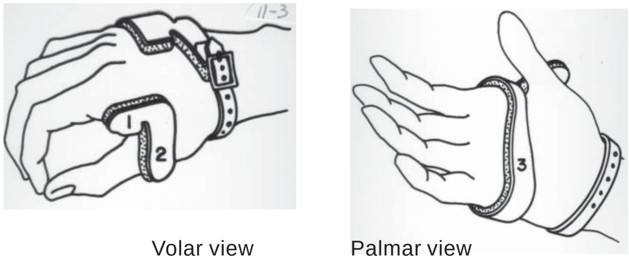


Fig. 11-2. Basic hand orthosis with radial extension.



Volar view

Palmar view

Fig. 11-3. Short opponens hand splint; volar view (left) and palmar view (right): (1) radial extension; (2) opponens extension; (3) palmar arch. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

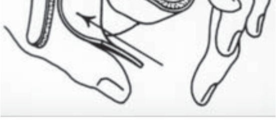


Fig. 11-4. Basic opponens splint with radial extension, opponens extension, and "C" bar (arrow). Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas;1965.

to move joints that are otherwise immobilized by the paralysis of the primary movers, and (*c*) use of external power.

If no possibility exists to transfer power from a strong muscle to a weaker muscle group, external power may be needed. Two sources of power commonly used are (1) compressed carbon dioxide (CO_2), or (2) an electric motor. The application of power is controlled by microvalves or switches that require minimal force to operate and can be positioned at any place where adequate muscle function exists. A small myoelectric signal may also be used through a microprocessor to proportionally control the external electric power.

Orthoses for Specific Functions

Basic opponens orthosis. This device substitutes for opponens and abductor pollicis brevis functions as they occur in median nerve lesions. To substitute for the absent or paralyzed opposition of the thumb to the fingers, the basic opponens orthosis, made of aluminum, is equipped with an opponens extension (see Figure 11-3). The opponens extension slides the thumb, by means of pressure on the first metacarpal, into opposition to the index and third finger pads. However, it does not move the thumb out of the palm. Therefore, grasp is only possible in the presence of function of the abductor pollicis brevis, which abducts the thumb perpendicular to the plane of the palm. If this muscle is weak or absent, another addition to the opponens splint (Figure 11-4) is the so-called "C" bar, which keeps the thumb out of the palm of the hand. With these modifications, usually a palmar or fingertip prehension can be produced, as well as a 3-jawed chuck. These modifications would oppose the thumb to the tips of the index and third fingers. Thumb flexors or finger flexors must be available for grasp and the finger extensors are needed for release.

The same functional orthosis can be produced by using plastics. By positioning the thumb against the fingers in the same fashion, an abducted and opposed thumb post is created (Figure 11-5). Many other modifications of this orthosis using heat-formed (formed when hot) plastics are frequently used in occupational therapy departments.[6] With further attachments to produce restraining forces, the basic opponens orthosis may be tilted off the hand, without added stabilization against the forearm. Because of this, a long opponens orthosis should be used (Figure 11-6).

Finger and metacarpophalangeal (MCP) extension attachments for conditions caused by radial

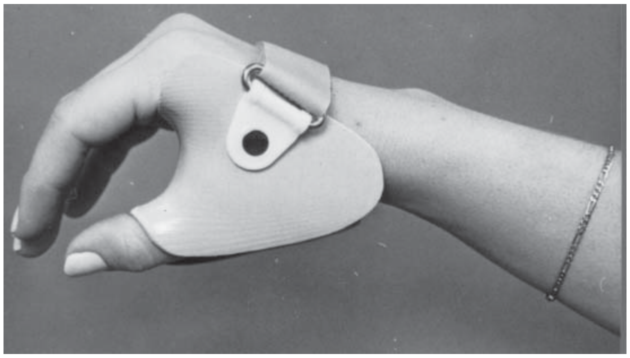


Fig. 11-5. Plastic basic opponens splint (Engen), keeping the thumb in opposition to the second and third fingers and abducted out of the plane of the palm.

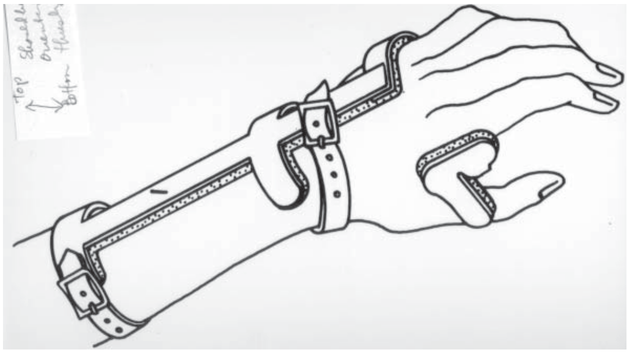


Fig. 11-6. Long opponens hand splint with forearm piece (1) and radial extension and opponens extension. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

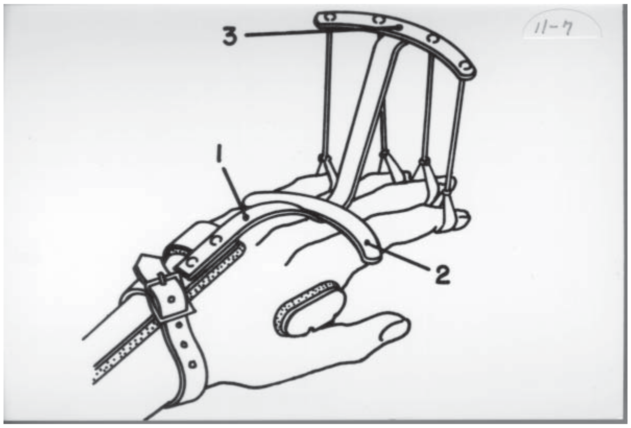


Fig. 11-7. IP extension assist with MCP stop: (1) allows detachment from the long opponens splint; (2) MCP extension stop; (3) outrigger with rubber band IP joint extension assist. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

nerve lesions. One attachment may be used to substitute for the interphalangeal (IP) joint extension. This orthosis (Figure 11-7) uses rubber band pulls with plastic sleeves to assist extension of the finger joints. Proximal or distal IP joint support is dependent on sleeve position. The bar just distal to the MCP joints serves to stop the MCP joints from going into hyperextension, due to the constant pull of the rubber bands. To prevent hyperextension of the IP joints, it is essential that the rubber band tension is finely tuned to provide just enough force to extend the fingers. Clearly, the force of the rubber band pull must be deducted from the finger flexor force, since the fingers have to work against the rubber band. Similarly, the extension of the MCP joints can be supported. For example, this orthosis should be used in cases of radial paralysis with good finger flexor strength, that is, intact ulnar or median nerve innervated flexors superficialis and profundus.

Long opponens splint with intrinsic bar (Figure 11-8). Normally, IP joints of the fingers are extended by the lumbricals and interossei, which also flex or stabilize the MCP joints; the extensor digitorum longus extends and, when unopposed, hyperextends the MCP joints. In the absence of these intrinsic functions, the extensor digitorum can be used to extend the IP joints for grasp release, provided that the intrinsic bar of the orthosis keeps the MCP joints in slight flexion. The unopposed action of the extensor digitorum would hyperextend the MCP joints associated with slight flexion of the IP joints.

The range of orthosis flexibility is illustrated by the following: a patient with a weakness of the extensor digitorum, with strong flexors digitorum

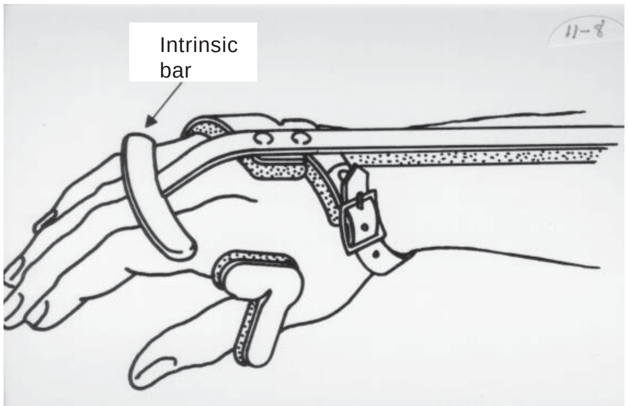


Fig. 11-8. Long opponens splint with intrinsic bar. Reprinted with permission from Anderson MH: *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

profundus and superficialis and available intrinsic musculature (such as palmar and dorsal interossei and lumbricals) has difficulty releasing the grasp. The patient may try to use the intrinsic muscles to extend the IP joints, at the same time, however, enacting unopposed maximal flexion of the MCP joints. If a wrist support orthosis is combined with a palmar piece that extends just beyond the MCP joints and limits flexion of these joints, the opening of grasp can be accomplished by the intrinsic muscles. These extend the IP joints, and the limitations of the MCP joint still allows a degree of functional grasp.

Opponens orthosis with holders for commonly used utensils. Multiple lesions may lead to conditions where this type of orthosis is needed. They may include upper motor neuron lesions, such as with SCI, or the combination of several peripheral nerve lesions. In these situations, adequate force of grasp and prehension are not available for effectively using these types of utensils (Figure 11-9). Attachments to an orthosis can hold such items as pencils, spoons, toothbrushes, and so forth.

Flexor Hinge Orthoses

Flexor hinge orthoses are primarily used in quadriplegia resulting from SCI.

Wrist extensor driven flexor hinge orthosis. Wrist extensor-driven flexor hinge orthoses[7] are shown in Figures 11-10 through 11-13. This orthosis stabilizes the thumb rigidly in opposition to the index and third finger. It stabilizes and immobilizes the IP joints of the second and third fingers in a semiflexed position for grasp and allows movement only in the MCP joints. As the fingers are flexed at these joints, the 3-jawed chuck pinch is produced, and, as they are extended, the grasp is released. The motor for this movement comes from the radial wrist extensors. The proximal part of the orthosis

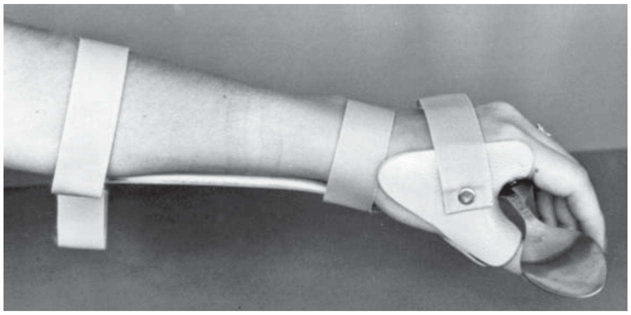


Fig. 11-9. Spoon fitted with a spring clip holder.

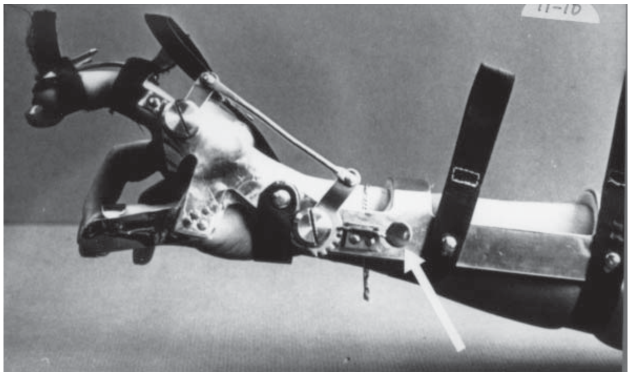


Fig. 11-10. Metal flexor hinge splint with adjustable ratchet controlling wrist angle at closing and opening: with fingers open; arrow indicates button to adjust ratchet.

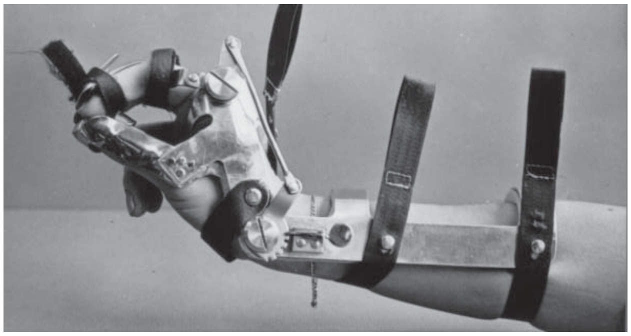


Fig. 11-11. Metal flexor hinge splint with adjustable ratchet controlling wrist angle at closing and opening: with fingers closed.

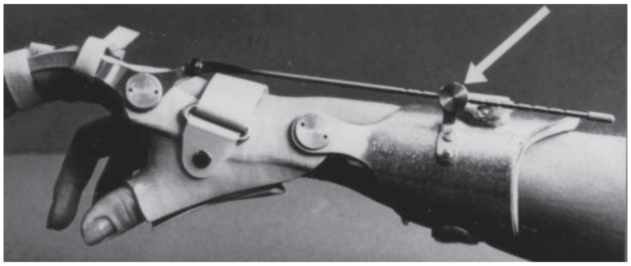


Fig. 11-12. Wrist-driven plastic flexor hinge splint with adjustable linkage across wrist to control extension and flexion angles at closing and opening (Engen); with fingers open, arrow indicates button to adjust the length of the linkage.

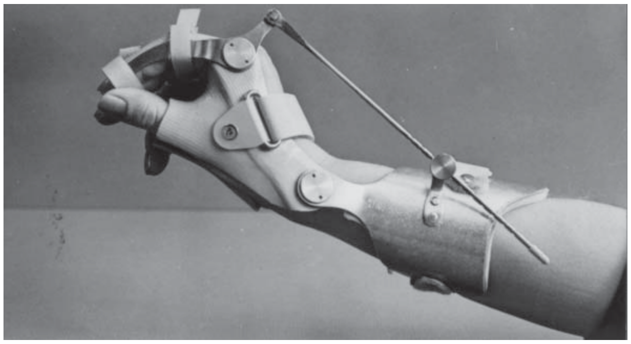


Fig. 11-13. Wrist-driven plastic flexor hinge splint with adjustable linkage across wrist to control angle at closing and opening (Engen): with fingers closed.

is connected with a hinge across the wrist, with the part of the orthosis extending over the forearm, as in the long opponens splint. The transfer of power from the wrist extensors occurs by metal rod linkage across the wrist, which produces grasp on extension of the wrist and release on flexion of the wrist (see Figures 11-10 through 11-13) This splint is especially useful for SCI patients who have some function at C5-C6, that is, the innervation of the radial wrist extensors, while the ulnar wrist extensors and the wrist flexors innervated by lower spinal segments are paralyzed. Therefore, grasp-release through wrist flexion is often provided by gravity or, on occasion, by rubber band pull that would oppose, in turn, the grasp movement produced by wrist extension. Because the radial wrist extensors receive the innervation from a higher level of spinal cord innervation, they are often stronger than the ulnar wrist extensors. Thus, a strong tendency exists to drive the wrist into radial deviation as it is extended. To prevent binding of the joint at the wrist, a flexible portion is incorporated into the orthosis at the wrist to allow this radial deviation to occur. Use of all flexor hinge-type orthoses necessitates some protective sensation between the thumb and index finger.

Shoulder harness driven flexor hinge orthosis. This orthosis is used in the presence of complete paralysis of the hand. Necessary physiologic conditions are some protective sensation in the fingertips and the voluntary ability to reach so that the hand can be positioned for grasp. Shoulder motion pulls the cable out of the housing and transfers the power from the shoulder musculature to the orthosis to release the grasp. Closure of the grasp is produced by rubber band or flexor spring pull (Figure 11-14). Since the rubber band would pull the fingers together all the time and, therefore, produce an ischemic necrosis of the tips, a mechanical release is provided which can stop the finger flexion with the fingertips apart. A quick pull release by the harness would overcome the stop and close the fingers.

External power driven flexor hinge orthoses.[8] The two sources of external power commonly used in flexor hinge orthoses are electrical motor drive or compressed carbon dioxide with artificial muscle (McKibben) as the motor. The electrical drive can be controlled by a switch, which the patient may activate by any muscle available. It can also be activated by an available, although weak, electromyographic signal of a voluntarily controlled muscle. By way of a microprocessor, this signal can proportionally control the motor drive.

The compressed CO_2 is provided in a metal cylinder. The gas is used to inflate an artificial muscle (McKibben), which consists of an inner rubber tube and an outer helically woven stocking. As the rubber tube is inflated and expands, the sleeve shortens in a similar fashion as a muscle. Inflation of the muscle produces a 3-jawed chuck grasp. The grasp release after deflation can be produced by gravity, spring, or rubber band pull. Inflation and deflation of the muscle are controlled by microvalves, which can be manipulated by any minimal voluntary power available any place. Like the other models of flexor hinge splints, the basic mechanical design remains the same, and this power driven orthosis should be used only in the presence of some protective sensa-

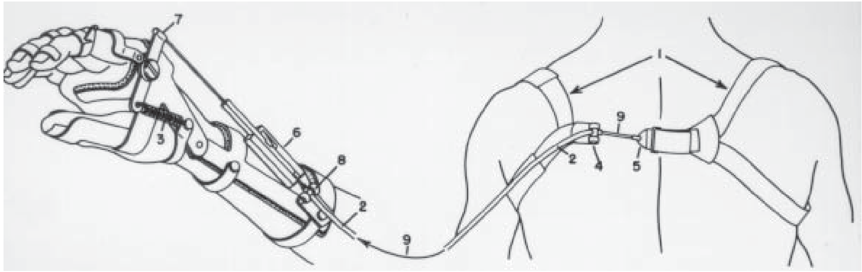


Fig. 11-14. Shoulder-driven flexor hinge splint. 1 = "butterfly" or shoulder-to-shoulder double loop harness; 2 = Bowden cable; 3 = flexor spring; 4 = leather retainer; 5 = stainless steel hanger; 6 = pressure relief control; 7 = finger piece operating lever; 8 = crossbar and anchor for cable housing; 9 = cable. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

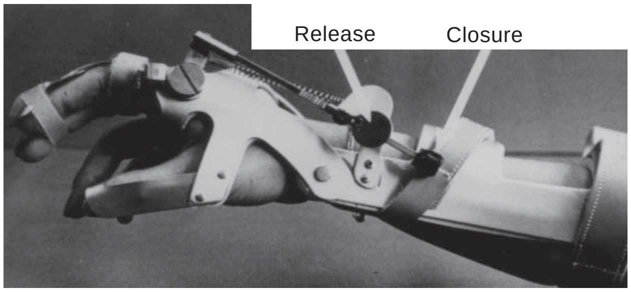


Fig. 11-15. Ratchet splint with push button for closing and pressure button for release; with fingers open.

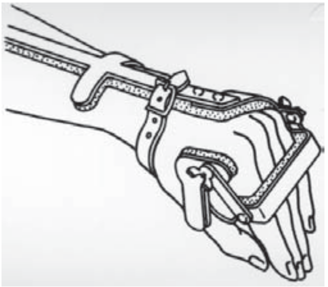


Fig 11-17. Adjustable MCP flextion control, "knuckle bender." Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

tion of the fingers and with a functional ability to reach. The power sources, electric batteries and gas cylinders, are usually attached to the wheelchair.

Passive prehension orthosis (flexor hinge splint, with ratchet control). This splint (Figures 11-15 and 11-16) allows a passive closing of the fingers by pushing a lever with a ratchet. Any force of prehension can be maintained, due to the ratchet stop. A button is pushed to open the ratchet lock, and grasp is released as a result of a spring pull. The force to push the ratchet lever to close the fingers has to be provided by the other extremity or by pushing against the lapboard or the wheelchair. This orthosis is commonly used for high level SCI patients who have no wrist extension and therefore cannot use the wrist-driven flexor hinge splint. Controlled prehension and release has also been achieved using functional electrical stimulation (FES),[9] although its use is uncommon.

Orthoses for Reduction of Contractures

Contractures can develop in cases of long-standing paralysis with partial immobilization of the joints of the hand and wrist, or as a result of prolonged casting. Contractures can limit either flexion or extension, depending on the position at which the joint has been immobilized.[10] In addition to physical therapy, a so-called "knuckle-bender" orthosis can be used to apply a long-term stretch to reduce the contracture (Figure 11-17).[11] There are many models constructed of metal, plastic, or springs, which include the knuckle-benders, or Bunnell splints, and allow endless varieties of positions for the fingers and wrist. These orthoses were frequently used for the Persian Gulf War casualties, and incorporated the wrist-hand orthotic base and outriggers with rubber bands for prolonged static stretch.

Interphalangeal Joint Stabilizers

If there is any instability of an abnormal IP joint, an IP stabilizer can be used to make the hand more functional by preventing movement at an IP joint (Figure 11-18).

Wrist Orthoses

Volar Wrist Flexion Control Orthosis

The volar wrist flexion control orthosis (cockup splint) is usually made out of plastic (Figure 11-

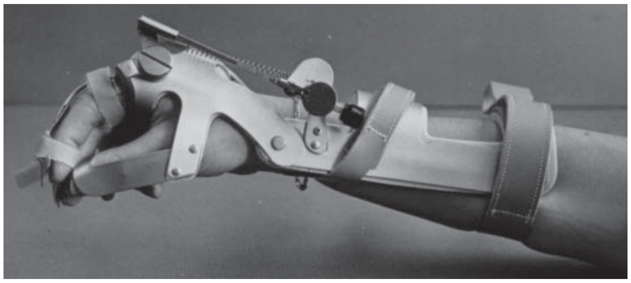


Fig. 11-16. Ratchet splint with push button for closing and pressure button for release; with fingers closed.

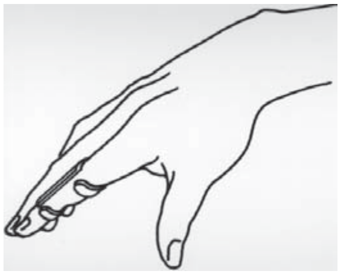


Fig. 11-18. IP joint stabilizer. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

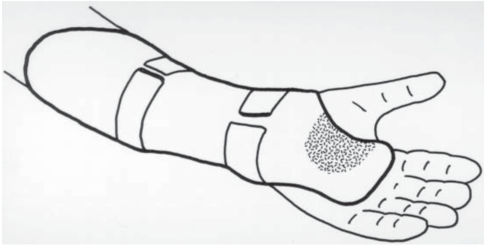


Fig. 11-19. Volar wrist flexion control orthosis, acrylic polyvinyl chloride.Photograph: Courtesy of DeRoyal LMB, San Luis Obispo, Calif.

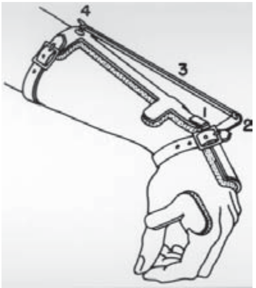


Fig. 11-20. Action wrist with dorsiflexion assist. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

19).[12,13] It consists of a rigid volar formed section, which continues into the palm. Straps over the dorsum of the forearm hold the orthosis in place. It supports the wrist in approximately 30° of dorsiflexion. In the presence of weak wrist dorsiflexion, the unopposed finger flexors pull the wrist into maximal flexion, and thereby allow maximal shortening of the flexor muscles. In this shortened position on the length-tension diagram, the strength of grasp is significantly reduced. This orthosis allows full grasp strength by limiting finger flexor excursion through immobilization of the wrist, and is used in cases of weakness of the wrist extensors.

Wrist Extension Assist (Action Wrist Extension with Dorsiflexion Assist)

A wrist extension assist orthosis consists of a forearm portion applied to the dorsum of the forearm with a hinge at the wrist. The portion of the orthosis over the hand distal to the hinge is similar in construction to the basic opponens orthosis, but without attachments. The spring or rubber band passing across the wrist dorsally assists the wrist extensor muscles. The force provided by the rubber band or spring extension combined with the remaining wrist extensor musculature strength must stabilize the wrist against the considerable forces of the finger flexors pulling the wrist into flexion, a condition which is rare. A prototype example is the long opponens splint with a hinge at the wrist and rubber band or spring extension assist (Figure 11-20). This orthosis can also be used for radial nerve injuries when it is combined with MCP extension assists.

Devices to Provide Reach

In many conditions, such as those caused by high level cervical spinal cord lesions, the patient may not have enough voluntary muscle control to place the hand in a position of grasp or to transfer objects, once grasped, from one location to another. At the same time, many of these patients are not ambulatory and are confined to a wheelchair. Therefore, the most common devices used for support of the arm, allowing some limited reach, are ball bearing mobile arm supports[14] and overhead slings, all of which are attached to a wheelchair.

Ball Bearing Mobile Arm Support

The support bracket for the ball bearing mobile arm support[15] (Figures 11-21 through 11-24) is attached to the steel tubing on the back of the wheelchair. The incline of the ball bearing in the sagittal plane can be adjusted; a swivel arm is inserted into this bearing. Another bearing is fixed at the distal end of this arm, into which the distal swivel arm is attached. Both ball bearings may have stops to prevent undesirable and uncontrollable motions. A trough that supports the forearm is attached to the distal swivel arm, and a dial attached to this trough keeps the forearm from slipping out of the trough support. The trough, in turn, is allowed to swivel up and down around a bearing located close to the center of mass of the forearm (Figure 11-25). By this design, which replaces active elbow flexion and extension, only minimal force is required to alter the balance of the trough and cause the hand to move down or up.

In addition, a mechanical guide can be installed so that the trough moves the hand down and, thus, into pronation for easy grasp. As the trough swivels the hand up, the hand is guided into supination for the feeding motion (Figure 11-26). As the trough moves the hand down, the guide moves the hand in pronation for easy grasp.

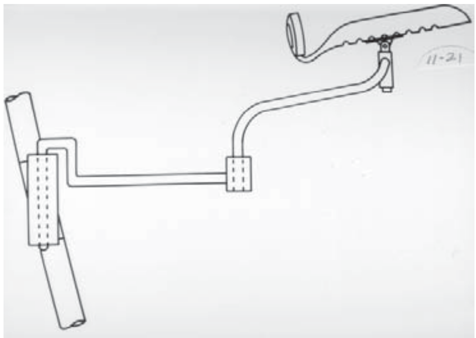


Fig. 11-21. Ball bearing mobile arm support, side view. Reprinted with permission from Wilson DJ, McKenzie MW, Barber LM. *Spinal Cord Injury: A Treatment Guide for Occupational Therapists*. Thorofare, NJ: Charles B. Slack; 1974.

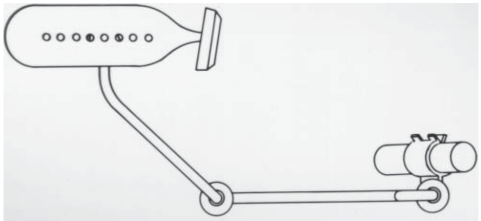


Fig. 11-22. Ball bearing mobile arm support, top view. Reprinted with permissin from Wilson DJ, McKenzie MW, Barber LM. *Spinal Cord Injury: A Treatment Guide for Occupational Therapists*. Thorofare, NJ: Charles B. Slack; 1974.

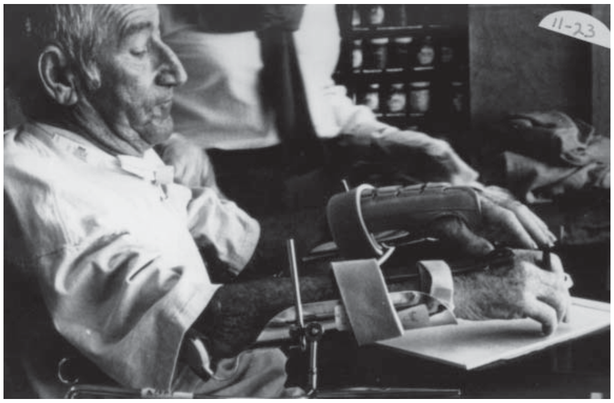


Fig. 11-23. Patient using ball bearing mobile arm supports and hand orthoses to write.

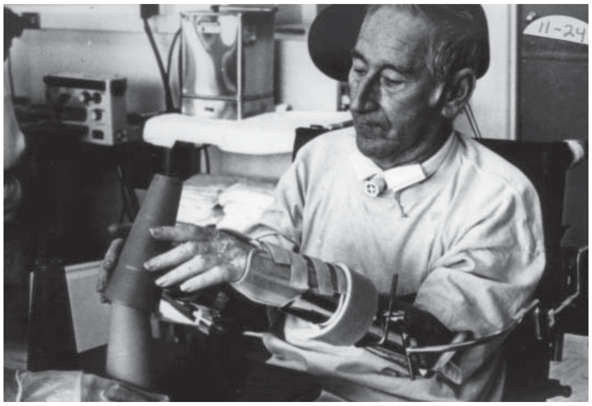


Fig. 11-24. Patient using ball bearing mobile arm supports and hand orthoses to stack coins.

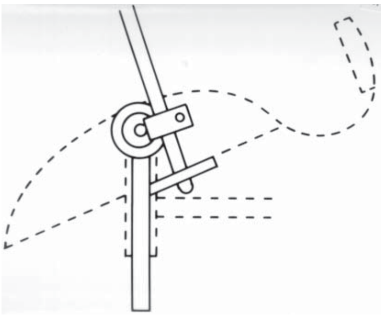


Fig. 11-25. Ball bearing mobile arm support, outside rocker arm assembly.

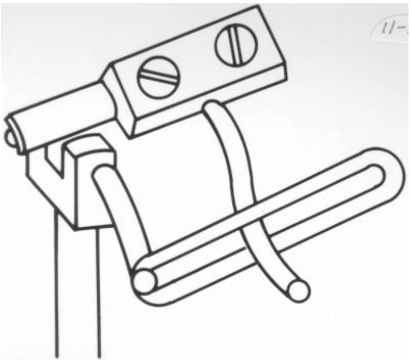


Fig. 11-26. Ball bearing mobile arm support, supinator assist.

If the ball bearing at the back of the wheelchair is adjusted so that the distal portion of the attached swivel arm points down and in a plane away from the body, gravity will extend the elbow and arm, moving the hand away from the body. If the rear ball bearing is adjusted so that the distal portion of the swivel arm is up, gravity will flex the elbow, and bring the hand close to the body. Therefore, the ball bearing support moves on the principle of an inclined plane. If the patient has musculature to pull the arm to the body, the plane inclines away from the trunk and helps to extend the reach. If the patient has no musculature to bring the hand close to the body or the mouth but has extensor musculature to push the hand and arm away from the body, the plane is adjusted to incline toward the body. In either case, if the adjustment is fine-tuned, only minimal forces are used to produce the desired movement. This type of substitution for a poor voluntary reach function is typically used in combination with some of the hand orthoses previously described.

The adjustment of this ball bearing mobile arm support, as well as the support trough for the arm, is critical for maximal utilization of the minimal voluntary force the patient will have available. Those who can benefit from this orthosis include patients with weak C-5 innervated muscles, brachial plexopathy, and SCI. All of these conditions occurred among Persian Gulf casualties.[16]

Forearm Orthosis with Friction Joints (Friction Feeder)

The same basic design, as in the ball bearing mobile arm support, can be used for a different purpose. The important design modification is that the ball bearing joints are replaced by joints with adjustable friction. The forearm orthosis with friction joints is able to control involuntary movements that often occur in various types of brain injuries. For instance, in conditions caused by cerebellar or cerebellar-pathway lesions, the orthosis is able to dampen ataxia and dysmetria, which enables the patient to get better controlled motion (Figure 11-27). In the same way, involuntary tremors, which often occur as a result of lesions of the basal ganglia, can be controlled.

Suspension Sling Arm Support

The suspension sling arm support uses essentially the same trough design as is used in the ball bearing mobile arm support, and allows swiveling of the hand down or up. The arm can also be supported (Figure 11-28) by a sling at the elbow and one at the wrist. The slings are suspended by a horizontal bar, which allows balance in favor of hand elevation or downward movement. This swivel bar is attached to the overhead support. In case of availability of muscles that depress the shoulder, it is sometimes useful to insert a spring between the overhead support and the swivel bar. The overhead support is attached by a metal bracket to the wheelchair. The patient with the least amount of voluntary force uses the trough suspension, while one with the most voluntary movement may use the sling suspension with the spring inserted to utilize the availability of shoulder depressors. With the straight, nonextendable suspension from above, these slings work on the principle of a pendulum. The upper extremity is supported overhead against gravitational forces (see Figure 11-28). Reaching movements can be accomplished by pushing the arm and hand forward or pulling them back. The arm follows the movement of a pendulum. There-

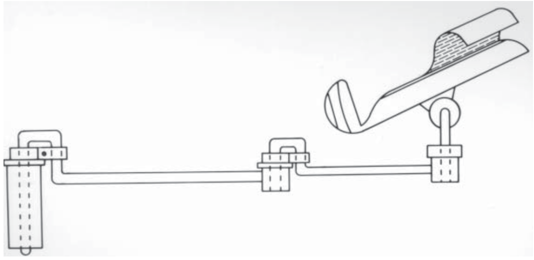


Fig. 11-27. Friction feeder, side view.

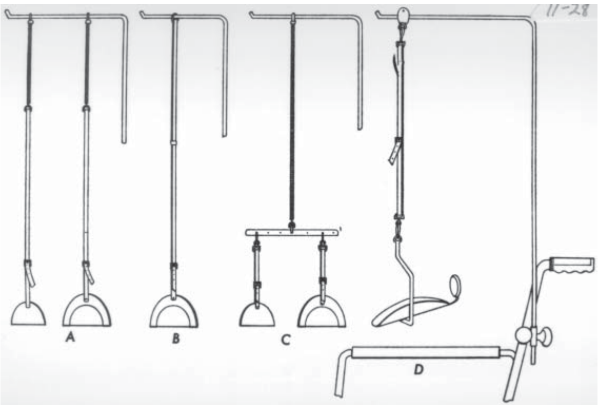


Fig. 11-28. Sling suspensions. **A**: double sling; **B**: single sling; **C**: sling with rocker arm; **D**; sling with rocker trough. Reprinted with permission from Long C, Schutt AH. Upper limb orthotics. In: Redford JB, ed. *Orthotics Etcetera.* 3rd ed. Baltimore, Md: Williams & Wilkins; 1986.

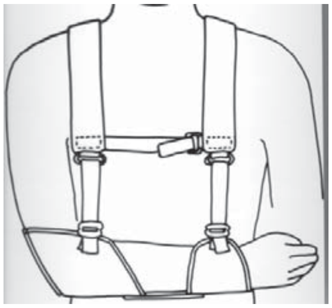


Fig. 11-29. Harris hemisling. Reprinted with permission from Brooke MM, de Lateur BJ, Diana-Rigby GC, Questad KA. Shoulder subluxation in hemiplegia: Effects of three different supports. *Arch Phys Med Rehabil.* 1991;72:582-586.

fore, some minimal lift and minimal force is required to push the arm forward or backward in relation to the rest position. To get adequate reach, these devices require significantly more voluntary muscle control than the ball bearing mobile arm support. In cases of brachial plexopathy, as well as SCI, it has been found that the suspension sling arm support increases shoulder range-of-motion, exercises the shoulder, and also provides a degree of early ADL independence.

Static Orthosis in Support of Shoulder and Elbow

Appliances commonly used on surgical services such as shoulder abduction orthoses, airplane splints, plaster casts, and other immobilization devices will not be discussed in this section.

Different types of shoulder sling supports are used in conditions which result from lack of voluntary control or reduction of muscle strength or tone of the deltoid or other glenohumeral joint musculature.[17-20] For instance, in central nervous system lesions that lead to hemiplegia, subluxation of the humeral head out of the glenoid of the scapula may occur from lack of shoulder support against gravity; thus, subluxation may be associated with pain and other complications. In a recent evaluation[21] of sling supports of various designs, the Harris hemisling (Figure 11-29) was found by radiograph measurements to give the best vertical correction, whereas the Bobath sling was not as effective and distracted the humeral joint horizontally. Sling designs similar to the Harris hemisling, such as the multiple and single strap designs (Figure 11-30), may be used in support of the forearm in the absence of elbow flexors. Other slings, such as the vertical arm sling, may support the arm, but allow elbow extension. Similar systems have been reviewed by Wynn Parry.[22,23]

Williams and colleagues[24] compared hemiparetic patients with shoulder subluxation treated with a Bobath shoulder roll to patients with the same condition treated with the Anderson shoulder ring. There was no difference between the two methods of management. However, greater subluxation occurred without any treatment.[24] Orthotic shoulder stabilizers for trapezius and serratus anterior weakness, stabilizing the shoulder, have been recommended by Villanueva[25] and by Truong and Rippel.[26]

Functional Orthoses for Shoulder and Elbow

Functional bracing of the shoulder and elbow is limited, since isolated functional weaknesses are rare, and only a few conditions lend themselves to bracing these two joints in an ambulatory patient. Also, various surgical approaches can be used, such as fusion of the glenohumeral joint or muscle and tendon transfers. Patients with high level SCIs and more widespread functional loss are often not ambulatory; therefore, the sling suspension or ball bearing mobile arm support must be used. Brachial plexus lesions often result in both motor and sensory losses and, therefore, bracing might not be the correct solution in the absence of protective sensation.

Basic Support of Shoulder and Elbow Orthoses

Development of an adequate support for the attachment of shoulder and elbow orthoses poses basically the same problem as in the hand: the need of firm attachment to allow proper function.[27,28] The shoulder and shoulder girdle are highly mobile and

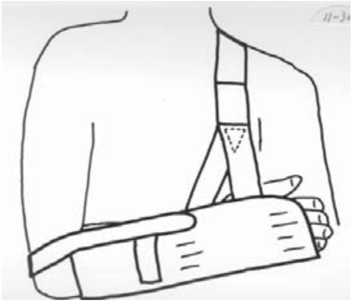


Fig. 11-30. Single strap design for shoulder support. Photograph: Courtesy of DeRoyal LMB, San Luis Obispo, Calif.

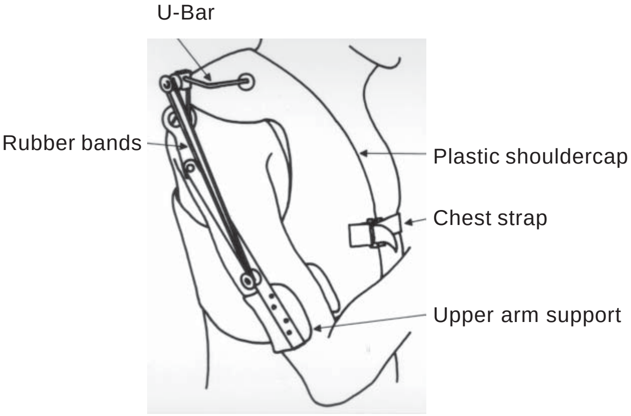


Fig. 11-31. Plastic shoulder cap design with rubber band shoulder flexion assist. Reprinted with permission from Anderson MH. *Upper Extremity Orthotics*. Springfield, Ill: Charles C Thomas; 1965.

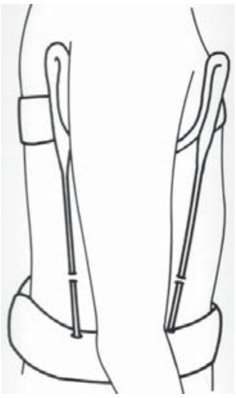


Fig. 11-33. Axillary shoulder suspension hoop.

do not readily allow direct application of orthoses[29]; therefore, a basic support structure has to be designed.

The basic support structure for the shoulder and elbow orthoses can be made of plastic in the form of a shoulder cap design that encloses half of the thorax below the axilla and covers the shoulder, and thereby allows freedom of shoulder motion. The cap is held in place by a chest strap or straps, and a U-shaped bar is attached to the cap of the plastic orthosis (Figure 11-31). Other modifying orthoses are ultimately attached to this U-bar. An alternative for this basic support structure is a pelvic band that encircles the trunk between the iliac crest and the greater trochanter. This band is rigid on the side of the orthosis and contains a soft closure on the opposite side. To this pelvic band, three rigid structures may be attached and held against the chest by a chest strap. These are the straight, the axillary, and the shoulder bypass suspension hoops (Figures 11-32, 11-33, and 11-34, respectively).

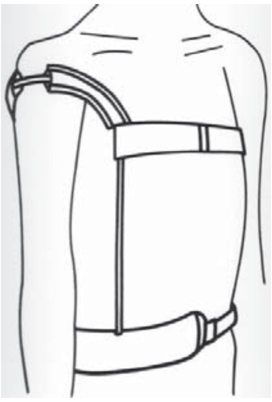


Fig. 11-34. By-pass shoulder suspension hoop.

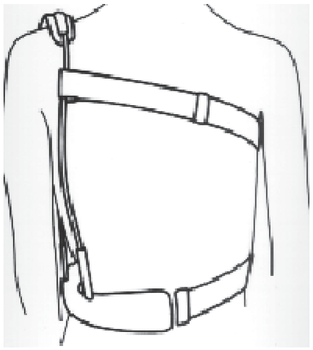


Fig. 11-32. Straight shoulder suspension hoop.

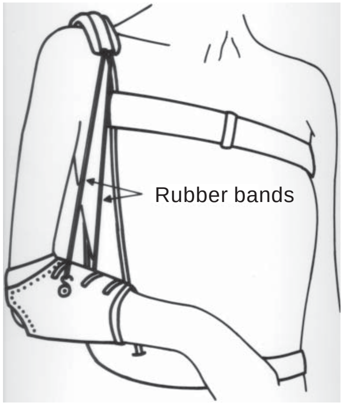


Fig. 11-35. Straight shoulder suspension hoop with forearm cuff elbow flexion assist.

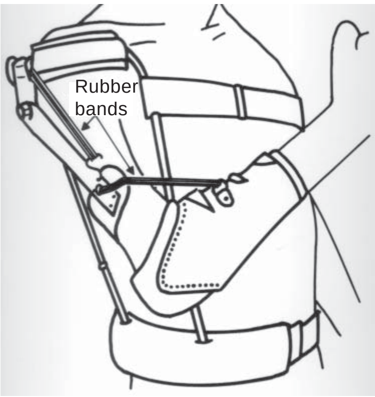


Fig. 11-36. Shoulder flexion assist with forearm cuff and elbow flexion assist.

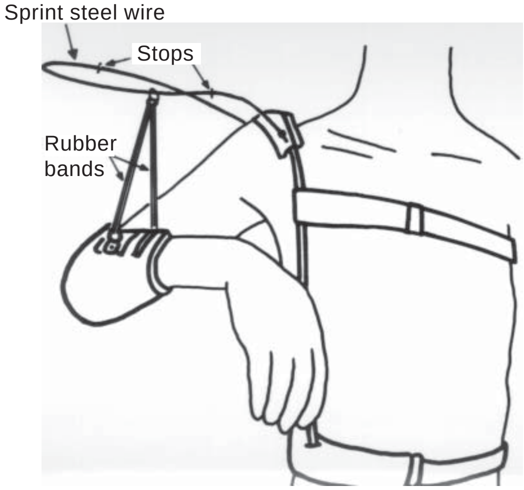


Fig. 11-37. Straight shoulder suspension hoop with abduction outrigger.

Orthoses are attached to these suspension hoops, as in the plastic design with the U-bar. As an example, Figure 11-35 shows a straight shoulder suspension hoop with an elbow flexion assist, that consists of a forearm cuff and rubber bands to support it. Figure 11-31 shows an orthosis with shoulder flexion assist that includes the rubber band. It is attached to the U-bar of the plastic base. Figure 11-36 shows an orthosis that is a combination of a forearm cuff elbow flexion assist and a shoulder flexion assist. As a basic support structure, the shoulder bypass suspension hoop was used. A final example of the approach to bracing the shoulder is shown in Figure 11-37. A shoulder bypass hoop is used for the attachment of an outrigger to assist shoulder abduction through a rubber band supported forearm cuff. Any movement against the rubber band support requires enough voluntary force to overcome the rubber band tension.

These examples show how components of available orthoses should be put together to fit the individual patient. Often, imagination is necessary to modify and even invent new designs to meet a particular patient need.

LOWER EXTREMITY ORTHOSES FOR NEUROMUSCULAR CONDITIONS

Ankle-Foot Orthoses

AFOs are the most commonly used braces for lower extremities. They are used in patients with TBI, SCI, and peripheral nerve injury. The AFOs substitute for loss of physiologic function, and additional walking aids are rarely needed unless there are other associated problems with a lesion that go beyond the paralysis or paresis of the leg and involve more widespread lower extremity groups, generalized spasticity, balance problems, or significant sensory feedback loss.

Components

AFOs can be made of metal or plastic. The metal orthoses discussed in this section are constructed of uprights of steel or aluminum that are connected on top and posteriorly by a rigid metal padded calf band with a soft front closure (Figure 11-38). They are attached below by an ankle joint to a stirrup incorporated into the sole of the shoe. The stirrup is often combined with a rigid metal sole plate which is riveted to the stirrup and extends to the metatarsal head area (Figure 11-39). Commonly, stops are attached to the ankle joint (Figure 11-40). There may be a posterior (plantar flexion) stop to substitute for the foot dorsiflexors and support the toe during the swing phase. These stops can be made rigid by the insertion of a metal rod into the posterior channel of the ankle joint, which engages the flange of the stirrup at an angle adjustable with a set screw. A more substantial assist for weak dorsiflexors would be a spring wire dorsiflexion assist orthosis, where a spring can be used instead of the rigid metal rod (see Figure 11-40).

The anterior stop (dorsiflexion stop) uses an adjustable pinstop, where the pin engages an anterior flange in the stirrup at a set angle. This stop should be used only in combination with the sole plate to the metatarsal head area; a spring assist will be too weak. This stop substitutes for the foot plantar flexors acting during pushoff. Figure 11-40 shows a Becker type ankle joint. The same stops are also commonly used in a Klenzak type joint (see Figure 11-38). Plantar flexion and dorsiflexion stops can be used together in the so-called double-stopped joint (see Figure 11-40).[30]

Indications

Most conditions requiring AFOs are due to weakness caused by upper and lower motor neuron lesions. The brace will accommodate spasticity and muscle imbalance. Many of these patients are able to walk without the orthosis; however, they are in danger of falling either because they trip as a result of the toe drag, or because mediolateral instability causes them to turn an ankle and fall.[31] Reasons for applying orthoses are to (*a*) provide mediolateral stability, and substitute for the lack of pushoff; (*b*) minimize knee instability during the early stance and increase stability during the late stance; (*c*) provide toe pickup during the swing phase; (*d*) approximate a normal gait pattern and, thus, reduce energy expenditure; and (*e*) prevent the development of deformities by improper weight bearing on weakened muscles.

Biomechanical Function

The biomechanical function of AFOs will largely depend on their design, irrespective of whether they are plastic or metal. Therefore, the basic biomechanical functions will be discussed with the standard double upright metal orthosis with stops at the ankle as a prototypical model.

Mediolateral stability. The orthosis in Figure 11-38 usually provides adequate mediolateral stability unless there is a strong tendency at the ankle to invert or evert. To prevent inversion (varus) of the foot, a T-strap may be attached to the sole of the shoe at the level of the instep, passing inside the lateral upright of the AFO and over the lateral malleolus, and cinched outside around the medial upright (Figure 11-41). As a result, the protruded lateral portion of the ankle is forced in line with the shoe and foot below and the calf band above, correcting the inversion. This type of correction is often needed in hemiparesis produced by stroke or TBI.

In eversion deformity (valgus) of the foot, the T-strap is applied to the shoe directly below the me-

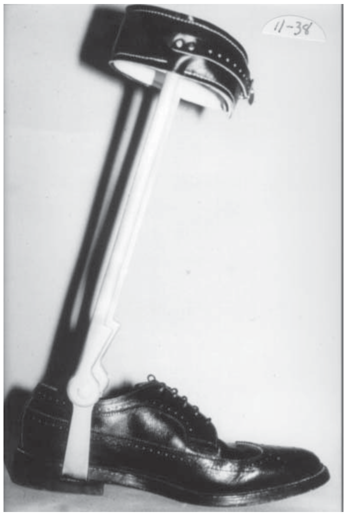


Fig. 11-38. Metal orthosis constructed out of bar stock metal uprights of steel or aluminum with Klenzak-type joint and posterior plantarflexion stop.

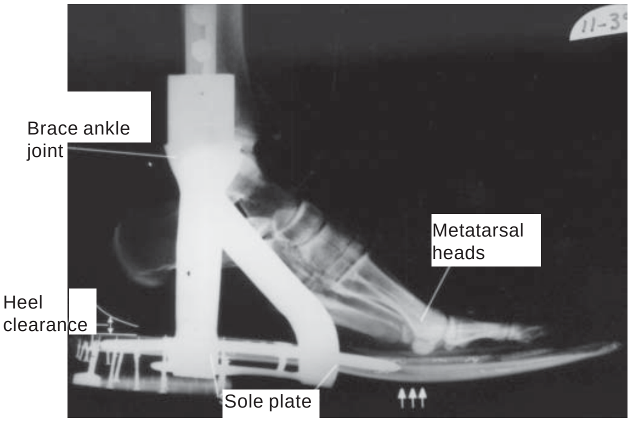


Fig. 11-39. Radiograph of a foot in an ankle-foot orthosis.

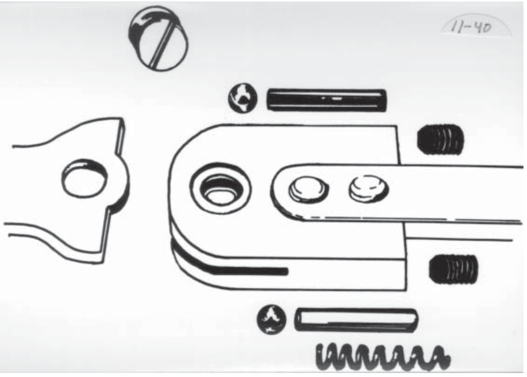


Fig. 11-40. Double-stopped ankle joint. The top stop limits dorsiflexion and the bottom stop limits plantar flexion. Reprinted with permission from Lehmann JF. Lower limb orthotics. In: Redford JB, ed. *Orthotics Etcetera.* 3rd ed. Baltimore, Md: Williams & Wilkins; 1986.

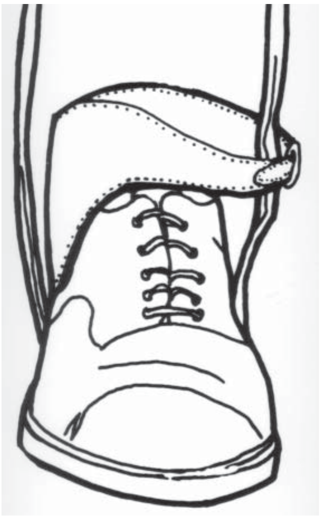


Fig. 11-41. Metal orthosis constructed out of bar stock metal uprights of steel or aluminum with Klenzak-type joint and medial T strap with posterior plantar flexion stop. Reprinted with permission from Lehmann JF. Lower limb orthotics. In: Redford JB, ed. *Orthotics Etcetera.* 3rd ed. Baltimore, Md: Williams & Wilkins; 1986.

dial malleolus and runs over the medial malleolus. It is cinched outside the AFO lateral upright to align the ankle with the shoe and foot below.

Posterior stops. The posterior stop substitutes for the weak ankle dorsiflexors during the swing phase to provide toe clearance. If there is an imbalance between the plantar flexors and the dorsiflexors, as with many conditions resulting from upper motor neuron lesions, a rigid pinstop may be needed to provide the force to pick up the toe against a strong spastic calf. If there is a flaccid paralysis, as happens with peripheral nerve injuries, a spring stop or spring wire brace may be adequate. Any of these posterior plantar flexion stops or springs supporting the toe during swing will produce more resistance to plantar flexion than the normal lengthening contraction of the dorsiflexors when the foot moves from heelstrike to the footflat position. Therefore, they all produce an increased bending moment at the knee during the heelstrike and early stance that the patient has to overcome by voluntary knee extensor musculature. The bending moment is caused when the action line of the ground reaction force (Figure 11-42) falls behind the knee and thus creates the bending moment. The ground reactive force line starts from the center of pressure, that is, the heel contact against the ground, while the patient rocks over the posterior extremity of the heel during the heelstrike phase. The amount and duration of bending moment at the knee depends on the amount of force in resistance to plantar flexion. Therefore, the greatest bending moment is created by the pinstop, rather than by spring assists.[32-34]

The duration and magnitude of the bending moment will also depend on the angle at which the pinstop engages the flange of the stirrup and stops plantar flexion. The more the foot is stopped in dorsiflexion, the greater the bending moment and its duration. Therefore, whenever a spring assist is adequate, a pinstop should not be used because the bending moment at the knee would be increased and would require more extensor muscle force to keep the knee from buckling. However, in special cases, where the gastrocnemius-soleus drives the foot into forceful plantar flexion, the pinstop must be used even though the voluntary knee extensor force may be marginal. In these cases, the adjustment of the stop will be changed to allow the foot to be stopped in more plantar flexion. This will reduce the bending moment at the knee and its duration during heelstrike. However, if the posterior stop is adjusted to allow enough plantar flexion so that the bending moment at the knee is reduced to keep the knee stable, it is possible that this angle will not allow adequate toe clearance during the swing phase; therefore, another method must be used. The posterior pinstop will be adjusted to stop the ankle in more dorsiflexion, allowing adequate toe clearance. In this case, the orthosis can be modi-

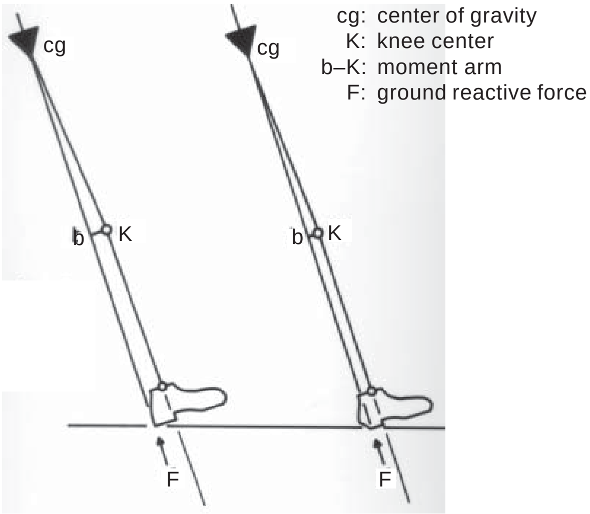


Fig. 11-42. Reduction of knee bending moment during heelstrike phase by heel cutoff. Reprinted with permission from Lehmann JF. The biomechanics of ankle foot orthoses: Prescription and design. *Arch Phys Med Rehabil.* 1979;60:200-207.

fied either by cutting off the posterior aspect of the heel at 45° (see Figure 11-42) or by inserting a cushion wedge into the heel. In both cases the location of the ground reactive force is moved forward. Thus, the extension of the force line comes closer to the knee, the moment arm gets smaller, and the bending moment is reduced so that the patient, in spite of marginal extensor musculature, can complete the heel strike phase without buckling and can adequately clear the toe during the swing phase of gait.

Anterior stops. The anterior (dorsiflexion) stop should use only a pin; no spring assist will be strong enough to assist with pushoff.[30] The dorsiflexion stop is used in combination with the sole plate riveted to the stirrup and extending rigidly to the metatarsal head area (Figure 11-43). As the center of gravity of the body moves forward, the ground reactive force line falls in front of the ankle. Dorsiflexion at the ankle is stopped; therefore, the foot pivots over the metatarsal head area, that is, the end of the sole plate, and raises the heel (Figure 11-44). This simulates pushoff by advancing the center of gravity of the body and pelvis upward and forward. As a result, the ground reactive force line is in front of the knee joint with the moment arm also in front (Figure 11-45). Therefore, a stabilizing extension moment is created in the latter part of the stance. This moment is of greater magnitude and longer duration if the dorsiflexion stop is adjusted in more plantar flexion. At the same time, the toe clearance is less.[31,32] Therefore, there is a trade-off between knee stability and toe clearance.

The extent to which these orthoses can restore the normal gait pattern and pushoff is shown in the example using the dorsiflexion stop with sole plate to restore pushoff in case of tibial nerve paralysis.[35] The normal timing of gait events is shown in Figure 11-46. The changes after a temporary tibial nerve block paralyzing the plantar flexors show that the heel comes off too late, lengthens the midstance, and shortens the pushoff phase. Restoration of normal timing of heeloff can be produced by an AFO with the anterior dorsiflexion stop adjusted to 5° of plantar flexion. The restoration is less complete with the orthosis adjusted to 5° of dorsiflexion. Figure 11-47 shows the normal progression of the center of pressure in relation to the ankle (black triangle curve). The center of pressure moves from behind the ankle, through the ankle, and then rapidly forward, in front of the ankle. This rapid forward movement is due to the gastrocnemius-soleus action resisting further dorsiflexion as the center of gravity of the body moves forward. Figure 11-47 shows that, in

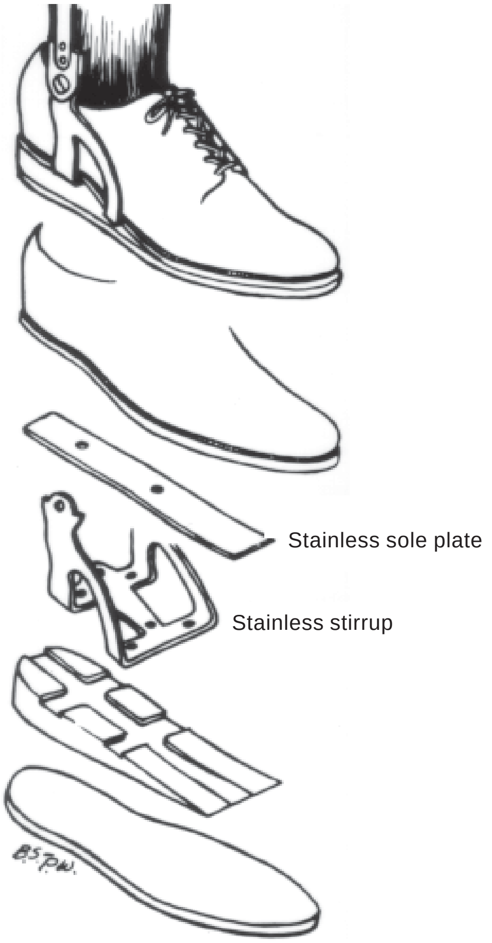


Fig. 11-43. Exploded view of stirrup, sole plate and shoe construction. Reprinted with permission from Lehmann JF, de Lateur BJ, Warren CG, Simons BC, Guy AW. Biomechanical evaluation of braces for paraplegics. *Arch Phys Med Rehabil.* 1969;50:179-188.

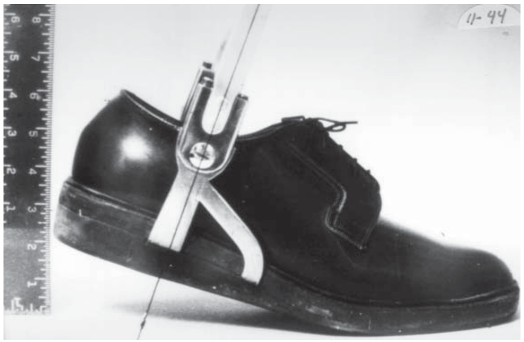


Fig. 11-44. Heel rise of brace with posterior and anterior stop and rigid sole plate. Reprinted with permission from Lehmann JF, de Lateur BJ, Warren CG, Simons BC, Guy AW. Biomechanical evaluation of braces for paraplegics. *Arch Phys Med Rehabil.* 1969;50:179-188.

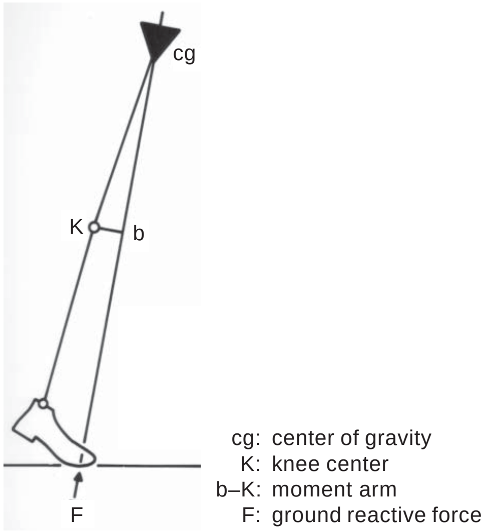


Fig. 11-45. Knee extension moment during pushoff. Reprinted with permission from Lehmann JF. The biomechanics of ankle foot orthoses: Prescription and design. *Arch Phys Med Rehabil.* 1979;60:200-207.

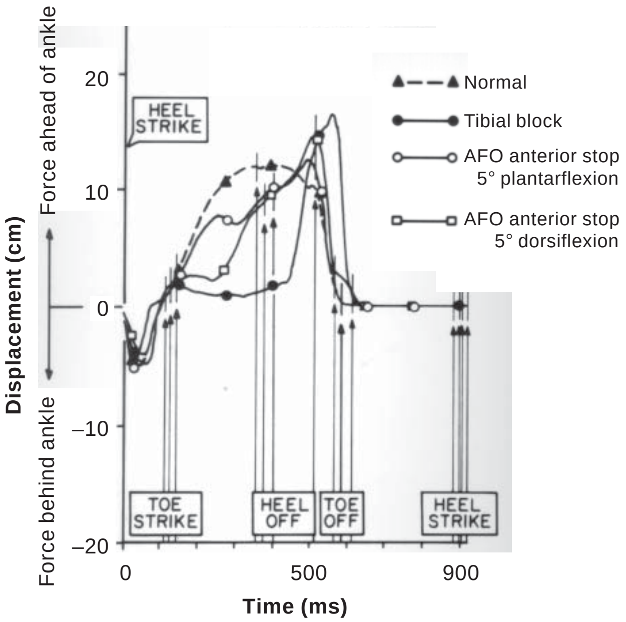


Fig. 11-47. Vertical force moment arm with respect to the ankle versus time. Curves represent the mean of six trials of one subject. Reprinted with permission from Lehmann JF, Condon SM, de Lateur BJ, Smith JC. Ankle-foot orthoses: Effect on gait abnormalities in tibial nerve paralysis. *Arch Phys Med Rehabil.* 1985;66:212-218.

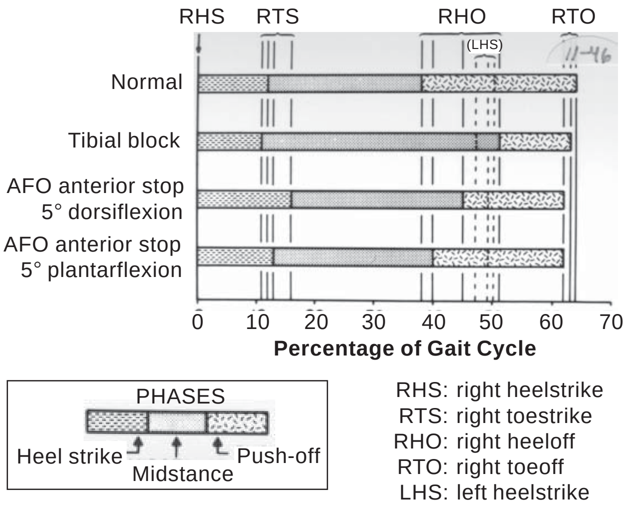


Fig. 11-46. Mean timing of gait events for six subjects. Reprinted with permission from Lehmann JF, Condon SM, de Lateur BJ, Smith JC. Ankle-foot orthoses: Effect on gait abnormalities in tibial nerve paralysis. *Arch Phys Med Rehabil.* 1985;66:212-218.

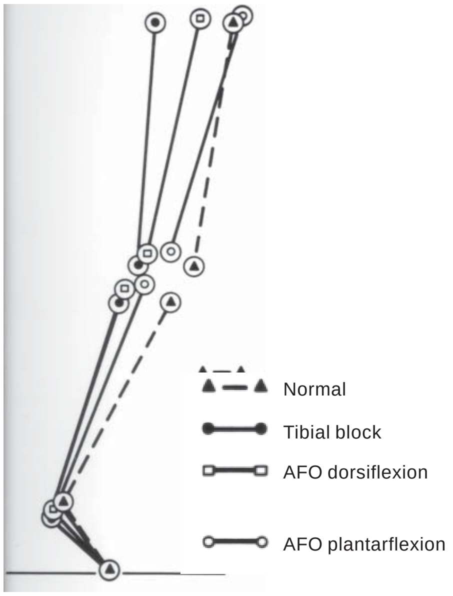


Fig. 11-48. Mean position of right lower limb at time of left heelstrike. Stick figures represent the means of six trials of one subject. Circles represent markers placed on the greater trochanter, lateral epicondyle, fibular head, lateral, malleolus, and fifth metatarsal head. Reprinted with permission from Lehmann JF, Condon SM, de Lateur BJ, Smith JC. Ankle-foot orthoses: Effect on gait abnormalities in tibial nerve paralysis. *Arch Phys Med Rehabil.* 1985;66:212-218.

the absence of gastrocnemius-soleus action, the patient holds back the center of gravity of the body. This is done to prevent the center of pressure and the force line from moving ahead of the ankle and producing unstable dorsiflexion (black dot curve). Forward movement of the center of pressure occurs only after the weight bearing has started on the opposite extremity. The open circles in Figure 11-47 show significant improvement when the adjustment of the dorsiflexion stop is at 5° of plantar flexion, with less improvement when the adjustment of the dorsiflexion stop is at 5° of dorsiflexion (open squares). Because pushoff is impossible with the paralysis of the tibial nerve and without braces, the forward movement of the center of gravity and the hip does not occur (Figure 11-48).

Full restoration of the forward movement occurs with the adjustment of the dorsiflexion stop at 5° of plantar flexion; partial restoration with adjustment at 5° of dorsiflexion. As a result of the lack of advancement of the pelvis and the lack of pushoff with tibial block,[35] the step length on the opposite side is reduced as compared with normal gait. The mean step length (in meters) during four conditions for the left (unblocked) side is shown below:

1. Normal (0.71)
2. Tibial block (0.55)
3. AFO 5° DF (0.66)
4. AFO 5° PF (0.63)

Conditions had a significant effect on step length ($p \leq 0.001$). Step length for the normal condition and for both AFO conditions was significantly greater than step length for the block condition. The normal condition step length is greatly improved by the AFO's dorsiflexion stop. The influence on knee stability by the nerve block is shown in Figure 11-49. Because the center of gravity of the body and, therefore, the ground reactive force line is held back at the ankle, the knee center moves well forward on the force line. This creates a large moment arm and a large bending (flexion) moment at the knee during stance. The bending moment is so great it requires considerable knee extensor force to prevent the knee from buckling. This is optimally corrected by the adjustment of the dorsiflexion stop to 5° of plantar flexion and also somewhat improved by the adjustment to 5° of dorsiflexion (see Figure 11-49). While this example shows that better correction of the paralyzed posterior calf can be achieved by using a dorsiflexion pinstop in combination with sole plate to the metatarsal head area, adjusted to 5° of plantar flexion, there is still a tradeoff with toe clearance during the swing phase. In this case, a compromise must be achieved.

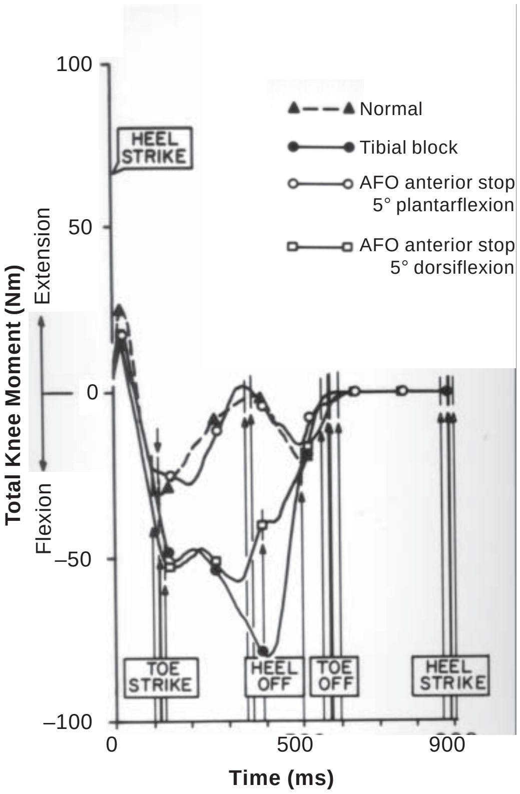


Fig. 11-49. Total knee moment versus time. Curves represent the mean of six trials of one subject. Reprinted with permission from Lehmann JF, Condon SM, de Lateur BJ, Smith JC. Ankle-foot orthoses: Effect on gait abnormalities in tibial nerve paralysis. *Arch Phys Med Rehabil.* 1985;66:212-218.

Design and Suitability

To understand the biomechanical function of any AFO, the design must be studied to determine its suitability for a given patient. In addition, a manual test of the maximum plantar flexion and dorsiflexion force the orthosis can resist should be conducted to decide whether or not the orthosis is adequate to control the forces created by the individual patient. It is always desirable to use the correction with minimal force, since over-bracing usually makes it more difficult for the patient to walk and maintain knee stability. With this caveat in mind, the biomechanical function of two brace designs will be discussed.

A plastic ankle-foot orthosis (PAFO)[36,37] has been developed using lamination techniques (Figure 11-50). The rigid ankle of this orthosis is equivalent to a double pinstop ankle joint; the rigid extension of the plastic to the metatarsal head area is equivalent to the metal sole plate. The orthosis is highly ac-

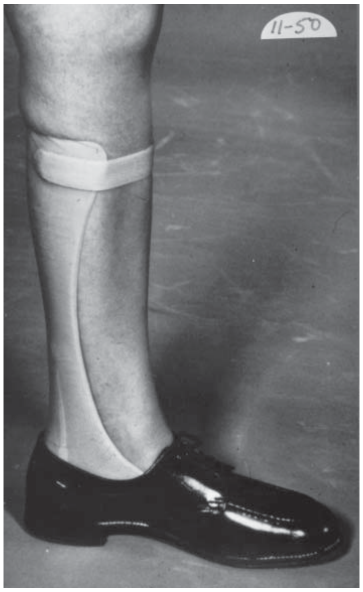


Fig. 11-50. Plastic laminated (Seattle) orthosis. Reprinted with permission from Lehmann JF. Biomechanics of ankle-foot orthoses: Prescription and design. *Arch Phys Med Rehabil.* 1979;60:200-207.

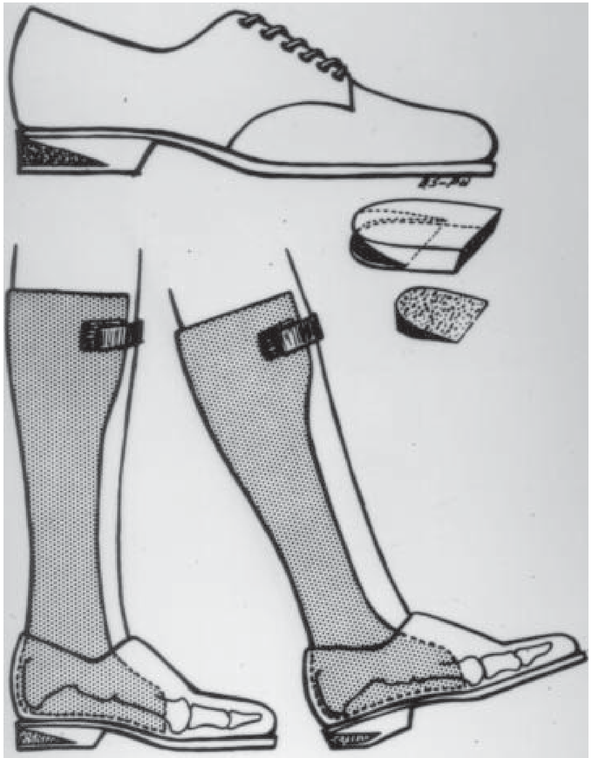


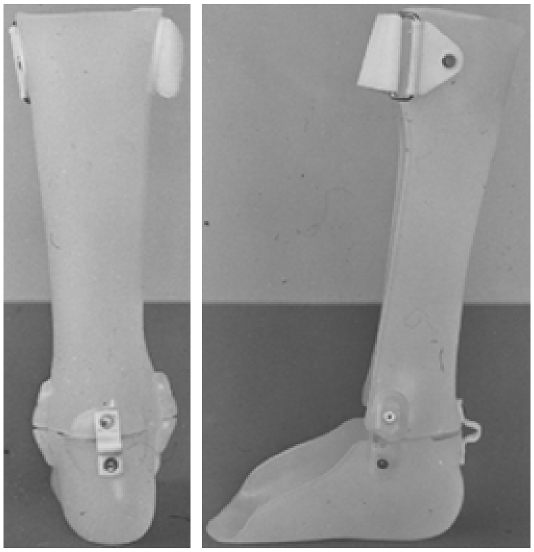
Fig. 11-51. Plastic laminated orthosis with heel cushion wedge, showing extension to metatarsal head area. Reprinted with permission from Lehmann JF. Biomechanics of ankle-foot orthoses: Prescription and design. *Arch Phys Med Rehabil.* 1979;60:200-207.

ceptable cosmetically (see Figure 11-50) and can be hidden under the stocking. However, it is critical that the ankle is fixed at the correct angle because of the tradeoff between knee stability and toe clearance. Because the trim lines (Figure 11-51) are in front of the ankle, the ankle is rigidly immobilized to provide maximum mediolateral stability. Shoes can be changed with this orthosis as long as the heel and sole height remain the same. The bending moment at the knee during early stance can be minimized by putting a cushion wedge into the heel or by a 45° cutoff of the heel (see Figure 11-51)

The manufacturing of this PAFO requires that a plaster cast be made with the correct angle at the ankle, with consideration given to the heel and sole height of the shoe. After this cast is removed, it is filled with plaster of Paris to produce a positive mold over which the plastic orthosis is laminated and then trimmed. If there is any mistake in casting, inversion, eversion, plantar flexion or dorsiflexion of the foot cannot be corrected, and the entire process has to be repeated. The use of wedges for correction of the ankle alignment typically results in an additional source of error.

This orthotic design has been advocated by Rancho Los Amigos Orthotic Department, but using heat-formed plastics with vacuum-molding techniques. Because the heat-formed plastics are thinned out most over the ankle area, the orthosis may become too soft to resist dorsiflexion and "open up" or "give way" at the ankle during pushoff. This does not provide adequate substitution for the forward and upward movement of the pelvis. To correct this problem, a thicker sheet of plastic should be used, or a boomerang-shaped carbon composite with beveled edges added over the positive mold.[38] This is so the hot plastic will flow over the beveled edges and incorporate these carbon composite pieces flush with the orthosis, thus providing a rigid equivalent to the dorsiflexion stop.

Whereas the PAFO described above is equivalent to a metal brace with an anterior and posterior rigid pinstop and a sole plate to the metatarsal head area, and does not allow any ankle motion, an articulated PAFO has been designed with functional adjustability of these stops. This orthosis consists of a separate portion for the foot and for the shank, connected by a hinge joint (Figures 11-52 and 11-53).[39] The angle at which dorsiflexion is stopped can be adjusted by the length of the posterior strap between the upper and lower parts of the orthosis.



Figs. 11-52 and 11-53. (**11-52,** left) Plastic articulated ankle-foot orthosis (AFO), rear view. (**11-53,** right) Plastic articulated AFO, medial view. **52** and **53:** Reprinted with permission from Lehmann JF, de Lateur BJ, Price R. Ankle-foot orthoses paresis and paralysis. *Phys Med Rehabil Clin North Am.* 1992;3:139-159.

The plantar flexion stop equivalent is provided by abutting the thickened edges of the upper portion and the lower portion posteriorly, so that they engage on plantar flexion. Trimming these edges produces more plantar flexion. Mediolateral stability is almost the same as in the other models of PAFOs.

Evaluation of AFOs by Biomechanical Principles

To evaluate an AFO, the sole and heel of the foot should be flat on the floor in quiet standing and during midstance. That it is flat can be best tested by using a thin piece of cardboard pushed between the sole of the shoe and the floor from the front, the back, and the lateral and medial sides. If the brace is tilted, the cardboard will move in too far from one or the other side. This fault can be corrected by bending the uprights or adjusting the angle at the ankle by the pinstops. In the PAFO, recasting is necessary. During the early stance (heelstrike phase), the knee should be observed for any tendency to buckle. If there is instability at the knee, the plantar flexion pinstop may be replaced by a spring, as in flaccid paralysis. If the pinstop is needed, it may be adjusted in too much dorsiflexion to create the bending moment at the knee.

During the latter part of the stance (pushoff), the knee should be observed for any tendency toward hyperextension. If that is the case, the pinstop should be readjusted at less plantar flexion or more dorsiflexion. During the swing phase, toe clearance should be checked for adequacy, and the stops readjusted if necessary.

In any orthosis that allows some plantar flexion and dorsiflexion, pistoning of the orthosis on the leg will occur unless the axis of the anatomical ankle (talocrural joint) is in the same location as the axis of motion in the orthosis. Therefore, it is desirable to line up the axis of the ankle joint of the double upright metal orthosis to coincide with the axis of the anatomical ankle, identified best by connecting the two tips of the malleoli (Figure 11-54).[40] In PAFOs with motion at the ankle, pistoning may be unavoidable except in the VAPC shoe clasp orthosis (Figures 11-55 and 11-56) where the pistoning of the posterior plastic rod is absorbed in a sleeve of the calf band.

In conclusion, the plantar flexion stop should use the minimum force required to prevent plantar flex-

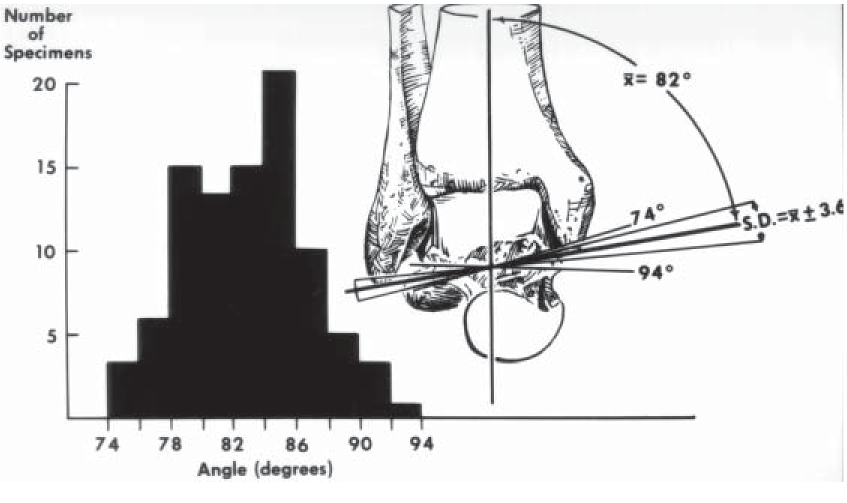


Fig. 11-54. Variations in angle between midline of tibia and empirical axis of ankle. The histogram reveals a considerable spread of individual values. Reprinted with permission from from Inman VT, Ralston HJ, Todd F. *Human Walking*. Baltimore, Md: Williams & Wilkins; 1981.

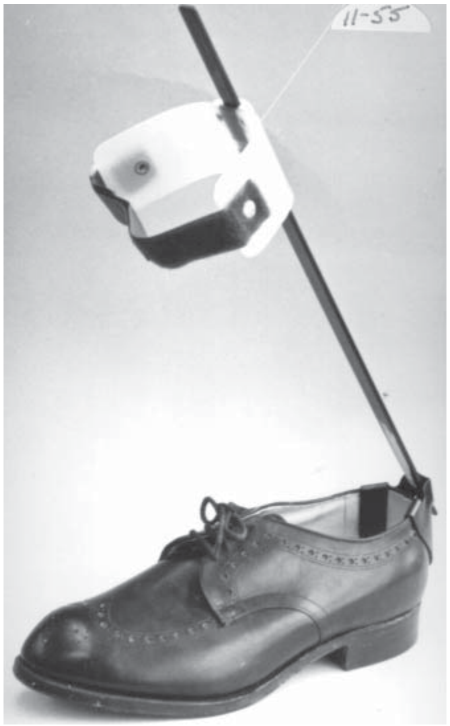


Fig. 11-55. The VAPC shoe clasp orthosis. Reprinted with permission from Lehmann JF, de Lateur BJ. Lower extremity orthotics. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* 4th ed. Philadelphia, Pa: WB Saunders; 1990.

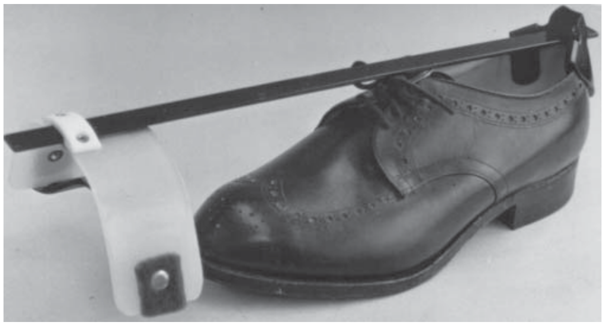


Fig. 11-56. The VAPC shoe clasp orthosis showing no resistance to dorsiflexion. Reprinted with permission from Lehmann JF, de Lateur BJ. Lower extremity orthotics. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* 4th ed. Philadelphia, Pa: WB Saunders; 1990.

ion, which results in toe drag. To minimize the bending moment at the knee that must be overcome with a voluntary effort, if adequate, a spring should be used. If a plantar flexion stop (posterior stop) is needed to provide the adequate forces against the plantar flexion during swing, a tradeoff exists between the amount of toe pickup and knee instability during the early stance at heelstrike. The less the stop is fixed in dorsiflexion and the more it is fixed in plantar flexion, the greater the knee stability at heelstrike. In the clinical setting, posterior stops should be used to provide minimum yet safe toe clearance during the swing phase and at the same time avoid unnecessary knee instability during heelstrike. Also, the anterior stops (dorsiflexion stops) should help support the knee during the latter part of the stance phase, especially in patients with marginal knee stability using the AFO.

Metabolic Requirements

Energy expenditure can be measured in terms of rate of energy consumption (cal/min/kg body weight) or in terms of efficiency of walking (cal/m walked/kg body weight). Table 11-1 gives the normal values.[41] In hemiplegic ambulation, the walking speed is markedly reduced. Therefore, the rate of energy consumption is close to the normal values and there is no excessive load placed on the cardiovascular and respiratory systems. On the other hand, the efficiency in terms of calories per meter walked per kilogram body weight is markedly reduced. The values are therefore increased (Table 11-2). As an example, a hemiparetic patient was studied walking without an orthosis, with a plastic orthosis, and with a double metal upright orthosis. Both orthoses had the same biomechanical functions. Therefore, the rate of energy consumption was the same in both cases. In addition, functional values, like the self-selected walking speed and the maximum walking speed, were the same but improved over the performance without an orthosis.

The energy consumption for any achievable walking speed was higher as compared with the normal curve, but slightly less than walking without orthoses. The patient had to be spotted continuously because of falling and twisting the ankle without an orthosis. With either orthosis, the patient could walk safely.

Assessing Different Designs for Patients' Specialized Needs

The biomechanical function of any orthotic design can be easily evaluated initially by looking at it. As an example, the Teufel orthosis, shown in Figure 11-57, is obviously a posterior plastic leaf spring orthosis, which provides toe clearance during swing, but is not likely to produce significant me-

TABLE 11-1
ENERGY REQUIREMENTS IN NORMAL AMBULATION*

Researcher and Date	N	Type of Disability	Speed (m/min)	Energy Expenditure (kcal/min/kg)	(kcal • 10^{-3}/m/kg)
Ralston, 1958[1]	19	Normals (M&F)	74†	0.058*	0.78
McDonald, 1961[2]	583	Normals (F)	80	0.067*	0.83
		Normals (M)	80	0.061*	0.76
Peizer, 1969[3]	?	Normals (?)	80‡	0.043‡	0.57
Corcoran, 1970[4]	32	Normals (M&F)	83†§	0.063*	0.76
Waters, 1976[5]	25	Normals (M&F)	82§	0.063*	0.77

*Calculated knowing kcal/meter, m/min, and weight.
†Most efficient speed of ambulation.
‡Approximated from a graph.
§Speed chosen by the subjects.
M: male
F: female

Adapted with permission from Fisher SV, Gullickson G: Energy cost of ambulation in health and disability: A literature review. *Arch Phys Med Rehabil*. 1978;59:125. Sources: (1) Ralston HJ. Energy-speed relation and optimal speed during level walking. *Int Z Angew Physiol einschl Arbeitsphysiol*. 1958;17:277-283. (2) McDonald I. Statistical studies of recorded energy expenditure of man. Part II, Expenditure on walking related to weight, sex, age, height, speed and gradient. *Nutr Abstr Rev*. 1961;31:739-762. (3) Peizer E, Wright DW, Mason C. Human locomotion. *Bull Prosthet Res*. 1969;10-12:48-105. (4) Corcoran PJ, Brengelmann GL. Oxygen uptake in normal and handicapped subjects, in relation to speed of walking beside velocity-controlled cart. *Arch Phys Med Rehabil.* 1970;51:78-87. (5) Waters RL, Perry J, Antonelli D, Hislop H. Energy cost of walking of amputees: Influence of level of amputation. *J Bone Joint Surg Am*. 1976;58:42-46.

TABLE 11-2
HEMIPLEGIC AMBULATION*

Researcher and Date	N	Type of Disability and Appliance	Speed (m/min)	Energy Expenditure kcal/min/kg	kcal • 10^{-3}/m/kg
Bard, 1963[1]	15	Hemiplegics	41*	0.044†	1.06
Corcoran, 1970[2]	15	Hemiplegics - no brace	42*	0.062	1.49‡
	15	Hemiplegics with plastic brace	49*	0.067	1.37‡
	15	Hemiplegics with metal base	49*	0.067	1.37‡

*Speed chosen by the subjects.
†Calculated knowing kcal/meter and m/min.
‡Calculated knowing kcal/min and m/min.

Reprinted with permission from Fisher SD, Gullickson G. Energy cost of ambulation in health and disability: A literature review. *Arch Phys Med Rehabil*. 1978;59:130. Sources: (1) Bard B. Energy expenditure of hemiplegic subjects during walking. *Arch Phys Med Rehabil.* 1963;44:368-370. (2) Corcoran PJ, Jebsen RH, Brengelmann GL, Simons BC. Effects of plastic and metal leg braces on speed and energy cost of hemiparetic ambulation. *Arch Phys Med Rehabil.* 1970;51:69-77.

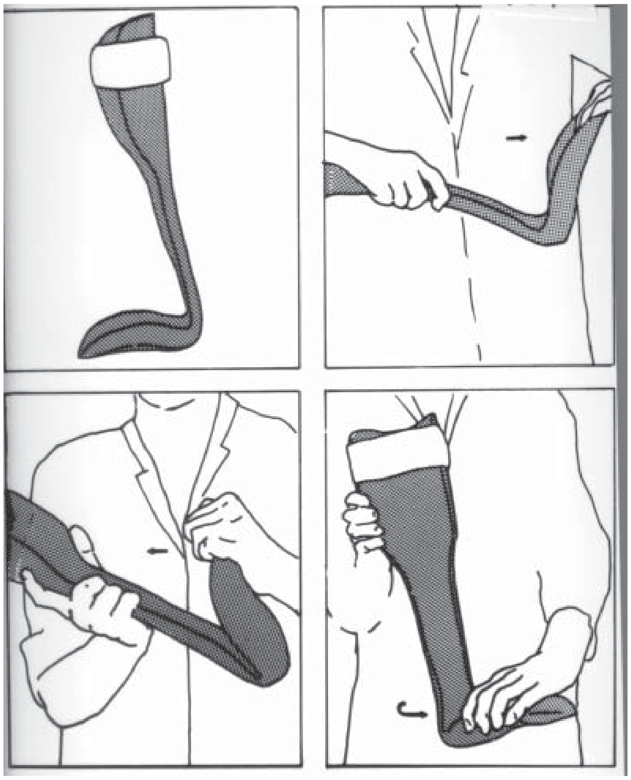


Fig. 11-57. The Teufel orthosis (top left); showing mediolateral twisting (top right); showing manual deformation into dorsiflexion (bottom left); showing manual deformation into plantar flexion (bottom right). Reprinted with permission from Lehmann JF. Biomechanics of ankle-foot orthoses: Prescription and design. *Arch Phys Med Rehabil.* 1979;60:200-207.

diolateral stability because it does not enclose the malleoli. This can be verified by twisting the orthosis to simulate the inversion or eversion of the foot, which the orthosis does not resist (see Figure 11-57, bottom right). Tests can measure the amount of force the orthosis provides against a calf that is driving the foot into equinus, as shown in Figure 11-57, top right. The lack of resistance to dorsiflexion demonstrates it is not a pushoff substitute (see Figure 11-57, bottom left). Figure 11-57 also shows that the axis of motion occurs in the orthosis at the posterior aspect of the heel and therefore some pistoning is unavoidable. A similar leaf spring design, the VAPC shoe clasp orthosis (see Figures 11-55 and 11-56), has essentially the same limitations, but it absorbs the pistoning motion by the insertion of a posterior plastic spring into a sleeve of the calf band. By pushing the orthosis into plantar flexion, an assessment can also be made of the force with which it will resist a spastic calf that is driving the foot into equinus position. Other orthoses can be tested as described in these examples, and based on this information, a customized orthosis can be prescribed for the individual.

Functional Electrical Stimulation of Peroneal Nerve

FES has been used in hemiparetic patients in an attempt to replace the AFO. Functional electrical stimulation was introduced by Liberson and coworkers[42] and was applied to the peroneal nerve in the area of the fibular neck to produce dorsiflexion and eversion during the swing phase of gait. Initially, skin electrodes were used, and the phasing of stimulation was controlled by switches incorporated in the shoe. Pulse duration of the current was between 20 and 33 microseconds with a repetition rate of 24 to 100 Hz, and intensity between 90 and 200 mA.

While this type of FES is rarely prescribed for permanent use, it is an extremely helpful tool when used during the recovery phase, for reeducation of the patient with hemiparesis resulting from brain injuries and stroke, and especially with patients who have paresis associated with decreased sensory feedback. While it produces a more normal gait pattern, it also produces a significant enhancement of sensory and proprioceptive feedback, including feedback from joint position sense and muscle contraction. If the proper level of stimulation at the peroneal nerve is used, it may produce not only dorsiflexion and eversion of the foot but also knee flexion and hip flexion during swing. For this purpose the stimulation level usually has to be increased to the nociceptive level. The whole system could be modified by implanting a receiving antenna under the skin of the anteromedial aspect of the thigh (Figure 11-58); a stimulus would be conducted to a surgically implanted electrode placed around the peroneal nerve. Stimulation would be activated by a transmitter antenna placed over the same area outside the skin where the receptor antenna was implanted. Through telemetry, the transmitter antenna receives the properly timed stimulatory signal, which is controlled by a transmitter, that in turn receives the signal from shoe switches. The transmitter and the battery pack are carried on a waist belt. The FES system was intended for long-term use. However, from the beginning, the indications for successful use of the system were fairly

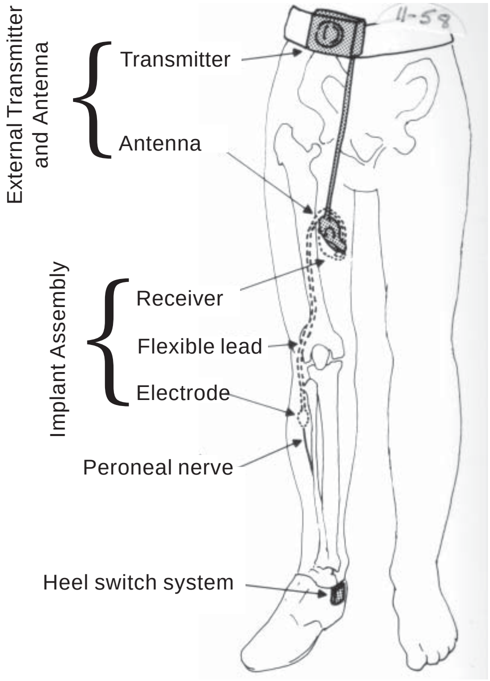


Fig. 11-58. Relative location of neuromuscular assist equipment on a patient with right side hemiplegia. Reprinted with permission from Lehmann JF. Lower limb orthotics. In: Redford JB, ed. *Orthotics Etcetera*. 3rd ed. Baltimore, Md: Williams & Wilkins; 1986; Redrawn with permission from Waters RL, McNeal D, Perry J. Experimental correction of foot drop by electrical stimulation of the peroneal nerve. *Arch Phys Med Rehabil.* 1975;53:276-281.

restrictive. Waters and Miller[43] found in their successful patients using FES that the patients had to walk faster than 25 m/min without any orthosis and needed good balance; and the system worked best for gait problems caused by foot drop.

One advantage of any electrical stimulation is that reciprocal inhibition of the gastrocnemius and soleus is produced when the peroneal nerve is stimulated. Significant problems with the FES system are that it must be surgically implanted, and if the wires break, repeat surgery will be necessary. Also, most patients prefer a simple PAFO that serves well and has fewer problems.

Knee-Ankle-Foot Orthoses

Knee-ankle-foot orthoses are commonly used for conditions caused by upper and lower motor neuron lesions.[44] Bilateral application is common in SCIs that produce upper motor neuron lesion, which cause spasticity, and lower motor neuron lesions of the cauda equina, which cause flaccidity. Unilateral use is more common when weakness of the quadriceps is combined with weakness around the ankle. The main function these orthoses provide, beyond those the AFOs provide, is unequivocal knee stability.[33,45] The reasons for application of these orthoses are to provide mediolateral stability at the ankle (as in AFOs); simulate pushoff by provision of an anterior dorsiflexion pinstop in combination with the sole plate to the metatarsal head area; and provide, during the swing phase, toe pickup by use of a plantar flexion stop at the ankle (as in AFOs).

Standard Components

The standard KAFO is constructed with double metal uprights, a knee joint (usually lockable with a bail), and a double-stopped ankle joint connected to a stirrup and sole plate at the metatarsal head area. The uprights are held together by padded rigid upper and lower posterior thigh bands and the calf band (Figure 11-59).[44] The relative depth of the upper thigh band to the lower thigh band will determine the position of the thigh and, therefore, the position of the knee in the orthosis. A deep upper thigh band with a shallow lower thigh band positions the thigh and knee in slight flexion, while a relatively shallow upper thigh band with a deep lower thigh band produces an extension of the knee. Tight and shallow refer to the distance of the posterior aspect of the rigid bands from the uprights.

Six common variations of the KAFO are used.[44] The orthoses vary significantly as to where the knee stabilizing force in front is applied. The corresponding counter forces are at the upper thigh band above and at the shoe below. Three brace configurations (see Figure 11-59) apply the stabilizing force through two straps to prevent the knee from bending in the brace. The first one uses the combination of a suprapatellar and patellar tendon strap, where both straps apply forces to very tolerant areas: quadriceps above the patella and the patellar tendon below. The second orthosis uses a soft closure of the lower thigh band in combination with the soft closure of the calf band. And, the third orthosis uses a combination of the lower thigh band closure and the patellar tendon strap. These three models have the advantage of distributing the knee stabilizing force over two straps, reducing the force per square centimeter on the skin.

The remaining three configurations use only one strap to apply the knee stabilization force: (1) with a suprapatellar strap, (2) with the patellar tendon

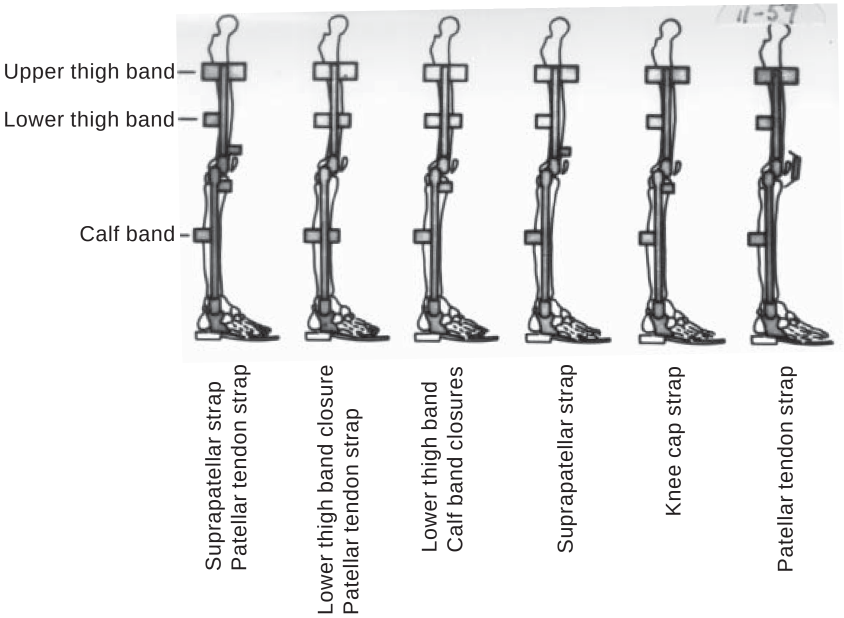


Fig. 11-59. Knee-ankle-foot orthoses: six common configurations. Reprinted with permission from Lehmann JF, Warren CG. Restraining forces in various designs of knee-ankle orthoses: Their placement and effect on the anatomical knee joint. *Arch Phys Med Rehabil.* 1976;57:430-442.

strap, and (3) with the knee cap strap, or spider. Note that the suprapatellar and the knee cap straps apply a single stabilization force above the knee joint. In the knee cap strap the force is applied to the patella, and the patella, in turn, applies the force through the patellarfemoral joint to the femur above the knee. All modifications of this orthosis have a soft closure of the upper thigh band in front.

These six configurations have been evaluated biomechanically, as to their effectiveness; a major objective of this evaluation was the reduction of the force applied to the skin by the knee stabilization bands. If the total stabilization force was measured irrespective as to whether it was applied by one or two straps, it was found that the highest force application was produced by the use of the lower thigh band and calf band closures because of the poor leverage of the forces applied far away from the knee center. This demonstrates that the stabilization forces should be applied close to the knee center.

Measurements of the stabilization force that keeps the knee from buckling in the brace reveal that if a few degrees of knee flexion are allowed in the orthosis, the force is doubled. The knee position can be controlled by the proper depth of the upper and lower thigh bands. It is also important that the brace be properly applied. Often the brace is applied while the patient is sitting, so that the stabilization straps are cinched too loosely; as the patient stands up, the knee bends in the orthosis and increases the forces against the straps. Proper recinching of the straps is necessary when the patient returns to a seated position. This clearly demonstrates that the brace must be designed and applied to keep the knee straight.

If the force required to stabilize the knee is measured at each strap, it will be found that the lowest force across the surface area of skin is produced by the combination of the suprapatellar and patellar tendon straps. These straps are close to the knee, distribute the forces between two straps, and apply the forces to very tolerant areas. Therefore, the minimal force across surface area is provided by a combination of the suprapatellar strap and patellar tendon strap. Since the patellar tendon strap applies the force to an extremely tolerant area, it also can be used alone, provided the strap is not placed over the shin.

A study of the shear forces acting on the knee ligaments in the orthosis in paraplegic swing-through ambulation showed that if an improper placement of the knee stabilizing straps was used these shear forces were excessive in amplitude and duration, with the femur shearing back on the tibia during the later part of the stance (Figures 11-60 through 11-62).[44] This shows that to avoid excessive knee shear, at least a major portion of the knee stabilization force should be applied below the knee.

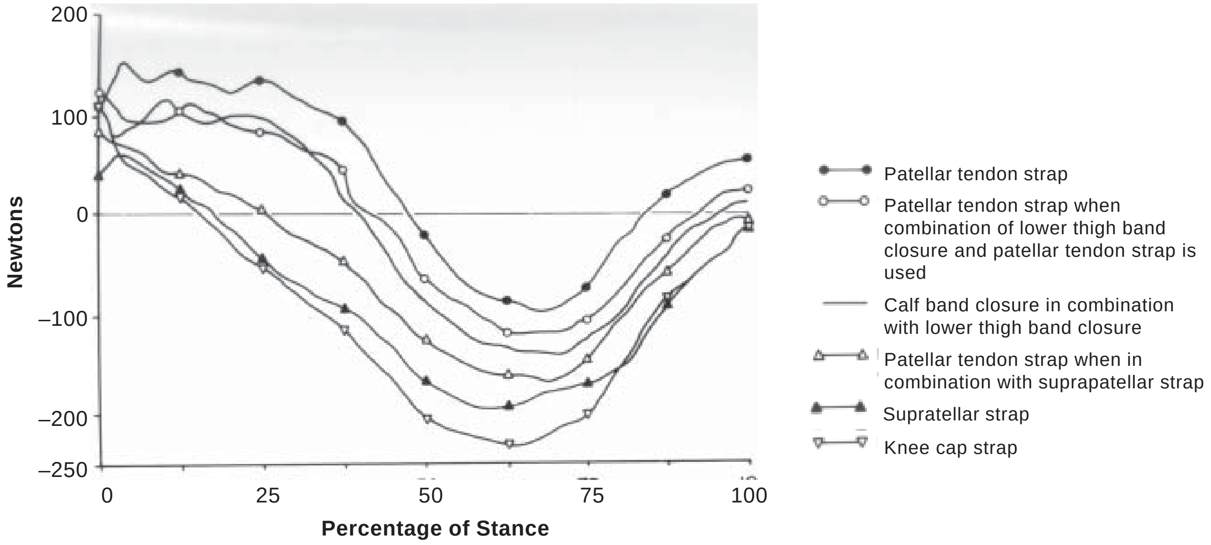


Fig. 11-60. Anatomical knee shear showing force interaction between femur and tibia. Positive values indicate the femur shearing forward on the tibia. Negative values indicate the femur shearing backward on the tibia. Reprinted with permission from Lehmann JF, Warren CG. Restraining forces in various designs of knee ankle orthoses: Their placement and effect on the anatomical knee joint. *Arch Phys Med Rehabil.* 1976;57:430-437.

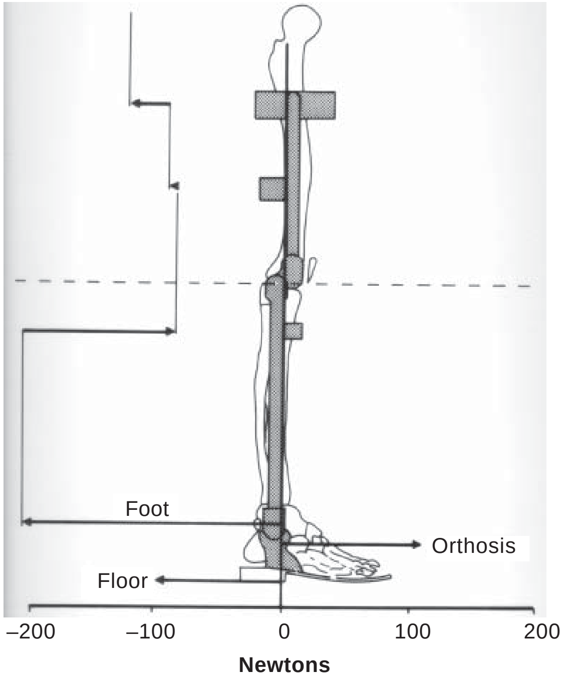


Fig. 11-61. Schematic representation of shear in the limb and orthosis during pushoff, the stabilizing strap below the knee. Reprinted with permission from Lehmann JF, Warren CG. Restraining forces in various designs of knee ankle orthoses: Their placement and effect on the anatomical knee joint. *Arch Phys Med Rehabil.* 1976;57:430-437.

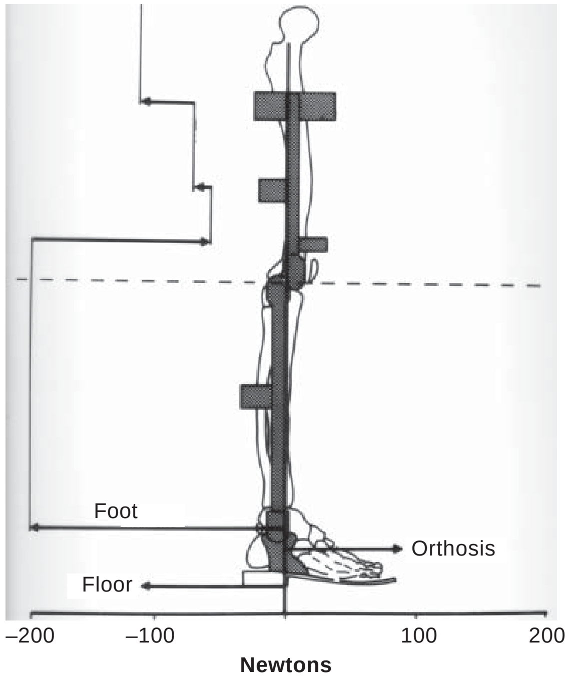


Fig. 11-62. Schematic representation of shear in limb and orthosis during pushoff with stabilizing force above the knee. Reprinted with permission from Lehmann JF, Warren CG. Restraining forces in various designs of knee ankle orthoses: Their placement and effect on the anatomical knee joint. *Arch Phys Med Rehabil.* 1976;57:430-437.

If the forces are measured against the edge of the upper thigh band when the orthosis is too long and does not allow enough clearance between the ischium and the upper thigh band with the hip extended, the forces during paraplegic swing-through ambulation are approximately 10 to 15 N/cm^3 (approximately 2,000 mm Hg). Therefore, abrasion and sores are produced because of this brace fault.[44] In conclusion, when the hip is fully extended, there should be at least two fingers width of clearance between the edge of the upper posterior thigh band and the ischium.

In summary, the orthotic designs of the biomechanically optimal KAFO should place knee restraining forces close to the knee, hold the patient's knee straight, distribute forces between straps, and apply the primary force below the knee. On hip extension, the upper edge of the posterior thigh band should adequately clear the ischium. In addition, the orthosis should be designed so that weight is reduced, donning and doffing are easy, free standing balance is possible even for a paraplegic patient with hands free, and energy expenditure is reduced.

Scott-Craig Orthosis

The Scott-Craig orthosis was designed to reduce the weight of the orthosis and to ease donning and doffing.[46,47] In this design, a posterior thigh band remains as the upper rigid connection between the two uprights of the orthosis. The lower posterior thigh band is eliminated. The bail lock at the knee is retained. The posterior calf band has been eliminated, but a rigid anterior shin piece not only connects the metal uprights below the knee but also supplies the knee stabilization force. The remainder of the orthotic design at the ankle and foot is the same as the standard orthosis. In evaluation studies[46] to determine if the rigidity of this design was adequate to handle stresses produced by paraplegic swing-through ambulation, it was found that a minimum of three rigid connections are required to hold the brace uprights together and prevent permanent deformation: the posterior upper thigh band above, the stirrup below, and one more rigid cross connection in the middle.

The Scott-Craig orthosis more than adequately fulfills the rigidity requirements; it also applies stabilization forces below the knee and, therefore, reduces knee shear. However, the original design of the knee stabilizing force should be modified to produce a plastic molded or padded piece that applies most of the force to the patellar tendon area in addition to the tibial condyles. This avoids development of skin sores in a vigorous walker, when only a rigid padded band over the shin is applied. Another modification required is the replacement of the eliminated posterior calf band by a soft closure to keep the orthosis from slipping from the conically shaped thigh, forward on the knee, when the patient sits down. With these modifications, the Scott-Craig orthosis improves donning and doffing and moderately reduces the weight of the orthosis.[46]

Metabolic Requirements

It is essential that metabolic requirements for patients using KAFOs, specifically for paraplegic or SCI persons, be reduced to a minimum. Paraplegic ambulation is limited because of the high rate of energy consumption when walking at a functional speed, compared to the mechanical ability of patients using a swing-through gait.[48-50] The features, which reduce energy consumption in any one of the orthoses already discussed, depend largely on the amount of substitution of pushoff the orthosis provides (see Figure 11-43).[51] The incorporation of a rigid anterior dorsiflexion pinstop at the ankle, in combination with the sole plate riveted to the stirrup and extending to the metatarsal head area, substitutes for pushoff. The standard orthosis and the Scott-Craig orthosis incorporate these features. During a swing-through gait, at the time when the two crutches are in front of the feet and the center of gravity moves forward, pivoting will occur at the end of the sole plate as dorsiflexion is stopped. Therefore, the heel rises and the center of gravity pathway curve is elevated at the lower end. As a result, the lift required for the clearance during swing is reduced.[46] Reduction of the amplitude of the center of gravity motion in the vertical direction saves approximately 30% of the mechanical work and metabolic requirements for ambulation. An orthosis not equipped with a dorsiflexion stop and sole plate does not provide substitution for pushoff and, therefore, does not save the 30% of energy consumption.

In conclusion, the addition to the KAFO of an anterior dorsiflexion pinstop in combination with the rigid sole plate to the metatarsal head area simulates pushoff, reduces vertical center of gravity excursions, and reduces mechanical work done and energy expenditure when the patient walks. This may prevent the patient from entering the anaerobic phase of metabolism, and, thus, increases walking endurance. Both the standard orthosis and the Scott-Craig orthosis provide this benefit.

TABLE 11-3

COMPARISON OF STANDING BALANCE

			Average Standing Balance/s		
Patient no.	Gender	Level of lesion	Post. Stop (n=5)	Post. and Ant. Stop (n=5)	t Value (*p*)
1	F	T12	1.55	300.0	1039.171 (0.0001)
2	F	T7-8	3.5	300.0	1228.081 (0.0001)
3	M	L2-3	3.6	246.0	8.602 (0.0001)
4	M	T4	1.58	8.9	1.218 (0.4872)
5	M	L3	40.25	80.15	1.640 (0.072)

Reprinted with permission from Lehmann JF, de Lateur BJ, Warren BS, Simons BC, Guy AW. Biomechanical evaluation of braces for paraplegics. *Arch Phys Med Rehabil.* 1969;50:187.

Standing Balance

The same KAFO features that produce substitution for the pushoff influence the standing balance. These are the anterior dorsiflexion pinstop in combination with the sole plate to the metatarsal head area, and the incorporation of a posterior pinstop at the ankle. In this brace configuration, an area of stable support exists within which the center of pressure may sway without imbalance of the patient. This support extends from the end of the sole plate in front to the posterior aspect of the heel, and from the side of one foot to the side of the other foot. In contrast, without the anterior dorsiflexion pinstop, the area of stable support extends only from the level of the ankle joint to the posterior aspect of the heels, which makes standing balance possible for only a few seconds rather than for minutes (Table 11-3).

The Use of Pelvic Bands with KAFOs

A pelvic band may be connected to the KAFO by a hip joint that may be locked through a drop lock. The pelvic band encircles the pelvis between the greater trochanter and the iliac crest. The front part of the pelvic band is rigid and padded, and the posterior part has a soft closure (Figure 11-63). Pelvic bands were expected to reduce excessive lumbar spine motion in ambulation that used a swing-through gait pattern. The band was also supposed to produce more standing stability, but a biomechanical functional evaluation has shown greater lumbar excursions with the pelvic band than without because of the restriction of motion at the hip for which the lumbar spine motion compensated. For the same reason, the mean stride length was reduced.[52] Due to the immobilization of the hip, a greater lift of the center of gravity of the body was required during swing. The time required for donning and doffing was almost doubled as compared with the standard KAFO. On the other hand, standing balance was slightly improved. Also, the uneven forward swing of the leg in paraplegic ambulation, which is caused by spasticity, was slightly improved. In most cases this could be overcome by training.

The pelvic band offers the slight advantage of controlling spastic lower extremities and improves standing balance. However, lumbar excursions and vertical excursions of the center of gravity are both increased. The step length is decreased. All these factors lead to an increased metabolic demand. Therefore, the pelvic band should be used only if there is a stringent reason for its application. A normal, swing-through gait pattern, where the patient has intact hip ligaments and no flexion contractures will allow the hip to be stable when the crutches are in front of the feet. In this position, gravitational forces extend the hip against the iliofemoral, pubofemoral, and ischiofemoral ligaments, around extension check ligaments, to stabilize the hip in extension. During the phase of the gait cycle where the crutches are behind the feet, arching of the back would place the center of gravity of the body above

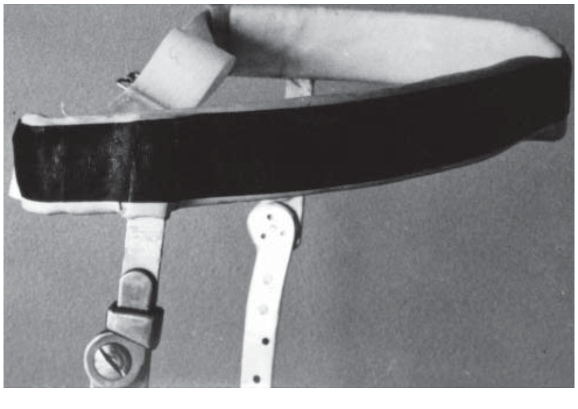


Fig. 11-63. The pelvic band. Reprinted with permission from Lehmann JF, de Lateur BJ, Price R. Knee-ankle-foot orthoses paresis and paralysis. *Phys Med Rehabil Clin North Am.* 1992;3:161-183.

TABLE 11-4

PARAPLEGIC AMBULATION

Researcher and Date	N	Type of Disability and Appliances	Speed (m/min)	Energy Expenditure kcal/min/kg	Energy Expenditure kcal • 10^{-3}/m/kg
Clinkingbeard, 1964[1]	4	Thoracic paraplegics	4	0.043	9.05
	3	Lumbar paraplegics	20	0.048	2.37
Gordon, 1956[2]	3	Thoracic paraplegics	27	0.090	2.44
	10	All paraplegics	27	0.086	2.32

Adapted with permission from Fisher SV, Gullickson G Jr. Energy cost of ambulation in health and disability: A literature review. *Arch Phys Med Rehabil.* 1978;59:130. Data sources: (1) Clinkingbeard JR, Gertsen JW, Hoehn D. Energy cost of ambulation in the traumatic paraplegic. *Am J Phys Med.* 1964;43:157-165. (2) Gordon EE, Vanderwalde H. Energy requirements in paraplegic ambulation. *Arch Phys Med Rehabil*. 1956;37:276-285.

and, therefore, the ground reactive force line behind the hip joint, thus extending it. This process locks the hip stably in extension against the ligaments. Therefore, there is no need for the pelvic band to stabilize the hip during the swing-through or swing-to gait patterns. In conclusion, the only advantages of the pelvic band would be some slightly better control of spastic lower extremities and improved standing balance.

Functional Use of Bilateral KAFOs in Paraplegia

The metabolic efficiency of paraplegic ambulation is poor (Table 11-4). To keep the rate of energy expenditure low enough to avoid entering the anaerobic phase of metabolism, which would reduce endurance, the paraplegic person considerably decreases walking speed.[43] Because the metabolic efficiency of walking also depends on the SCI level, it has been generally recommended that only persons with lower lumbar level lesions be provided with orthoses and trained for ambulation. A more functional way to look at this problem follows.

The demands for distance walked and speed of ambulation placed on the patient in his or her environment should be assessed irrespective of the level of the spinal cord lesion. If the paraplegic patient can cover the distance required and provide the speed needed, braces should be prescribed and training should be provided. For a long distance ambulator, the wheelchair should be considered since its metabolic requirements (Table 11-5) are less

TABLE 11-5

WHEELCHAIR LOCOMOTION*

Researcher and Date	N	Type of Disability and Appliances	Speed (m/min)	Energy Expenditure kcal/min/kg	Energy Expenditure kcal • 10^{-3}/m/kg
Hildebrandt, 1970[1]	30	Wheelchair-bound	67	0.037	0.47*
Glaser, 1975[2]	9	Normals	53	0.047†	0.89†
	9	Normals	70	0.056†	0.79†
	9	Normals	83	0.071†	0.84†

*Calculated knowing kcal/min and m/min

†Calculated from O_2 consumption

Reprinted with permission from Fisher SV, Gullickson G Jr. Energy cost of ambulation in health and disability: A literature review. *Arch Phys Med Rehabil.* 1978;59:131. Sources: (1) Hildebrandt G, Voigt ED, Bahn D, Berendes B, Kröger J. Energy costs of propelling wheelchair at various speeds: Cardiac response and effect on steering accuracy. *Arch Phys Med Rehabil.* 1970;51:131-136. (2) Glaser RM, Edwards M, Barr SA, Wilson GH. Energy cost and cardiorespiratory response to wheelchair ambulation and walking (abstract). *Fed Proc*. 1975;34:461.

than that of normal ambulation. For a high level of SCI, such as quadriplegia, even the use of a manual wheelchair may force the patient to use the anaerobic metabolism because of the small muscle mass involved in this activity. In those cases, an electric wheelchair should be considered.

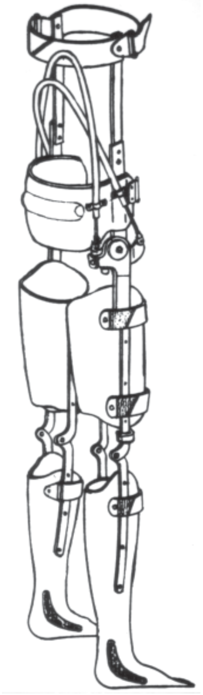
In conclusion, depending on the demands for ambulation placed on the SCI person by his or her environment and the ability to fulfill these requirements, the patient should be fitted and trained with orthoses or given a manual or electric wheelchair. This approach is in contrast to training for the use of braces and crutches solely based on the level of the spinal cord lesion.

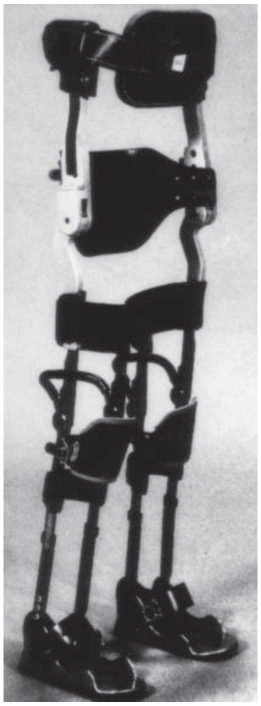
Reciprocating Gait Orthosis

Usually, walking speed is significantly limited with the reciprocating gait orthosis (RGO). In a study by Merritt[53] using speeds from 17 to 41 m/min, it was found that the oxygen consumption per kilogram of body weight per meter walked rose rapidly at speeds above 27 m/min when using the alternating gait pattern. By comparison, the Scott-Craig orthosis was more energy efficient when used with the swing-through gait at the same speed as the RGO. If the RGO was used like the Scott-Craig with a swing-through gait pattern, the energy consumption was the same in both cases. Thus, subjects using RGOs with reciprocating gaits have higher energy expenditures (VO_2/min) and poorer energy efficiencies (VO_2/m) than when using standard orthoses with a swing-through gait. Therefore, long sustained ambulation at functional speeds is more difficult than with the standard or Scott-Craig orthosis.

The RGO is commonly used for patients with SCIs, or those with cauda equina lesions. The orthotic design for the RGO is shown in Figure 11-64. To keep the weight of this orthosis within tolerable limits, a greater amount of plastic material is used compared to, for example, the AFO and the thigh cuff. In addition, one of the main features is that low friction Bowden cables are used, which transfer power from hip extension on one side to hip flexion on the other side, or power from hip flexion into hip extension on the opposite side. Thus, a reciprocating gait pattern can be achieved provided the patient has some hip flexion or hip extension power available at hip musculature. The orthosis braces not only the hip but also the trunk. Locking mechanisms for hip and knee can be added.

The development of the RGO has been based on the design of the hip guidance orthosis, which allows limitations to be set for hip flexion-extension and uses a rocking motion to get one leg in front of the other, thus producing a reciprocating gait pattern.[54,55] Originally, it was primarily designed for lower motor neuron lesions, such as meningomyelocoele and low level SCIs, that is, cauda equina lesions (Figure 11-65). Its modification for paraplegic persons is also known as the ParaWalker.[56]





Figs. 11-64 and 11-65. (**11-64**)The reciprocating gait orthosis. Photograph: Courtesy of Durr-Fillauer Medical, Inc., Chatanooga, Tennessee. (**11-65**) The ParaWalker. Photograph: Courtesy of ORLAU publishing. Oswestry, Shropshire, Great Britain.

Functional Electrical Stimulation for Bracing

FES of key muscle groups in proper sequence for walking has now been used in major SCI treatment centers.[57,58] The sequence is controlled either by the patient with switches, or by a microprocessor. For balance and stability, walkers or crutches are also used. The gait pattern is a reciprocal one. Common muscles stimulated are gluteus medius, gluteus minimus, gluteus maximus, iliacus, rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis.[59] Both surface electrodes as well as transcutaneous wire electrodes are used. The lack of sensation in the SCI patient is an advantage, as the level of current used for stimulation may be unpleasant. Further research in this area is required because the speed of ambulation with this type of brace is very

TABLE 11-6

ENERGY COSTS OF AMBULATION WITH FUNCTIONAL NEUROMUSCULAR STIMULATION

Subject	Trial Number	Distance (m)	Speed (m/s)	O_2 Consumption (L/min)	Energy Expenditure (kcal/kg/min)	Energy Expenditure (kcal/kg/m)	O_2 Debt (L)
1	1	58.5	0.32	1.36	0.089	4.74×10^{-3}	1.89
1	2	30.5	0.35	1.43	0.094	4.43×10^{-3}	1.62
1	3	29.6	0.17	1.57	0.099	9.84×10^{-3}	2.30
1	4	30.5	0.39	1.42	0.094	4.05×10^{-3}	1.33
1*	5	28.7	0.11	1.38	0.091	13.40×10^{-3}	1.62
2	1	58.5	0.16	1.64	0.103	10.98×10^{-3}	1.59
2	2	30.2	0.15	1.39	0.088	9.53×10^{-3}	1.73
2	3	39.6	0.13	1.52	0.101	13.13×10^{-3}	1.79
2	4	30.5	0.16	1.49	0.099	10.13×10^{-3}	1.59
2*	5	30.2	0.16	1.46	0.093	9.66×10^{-3}	1.80
3	1	58.5	0.56	1.48	0.093	2.77×10^{-3}	2.90
			$\bar{x} = 0.24$ m/s		$\bar{x} = 0.095$ SD = 0.005		

*No hamstrings or gluteals; subject 1: $\bar{x} = 0.093$, SD = 0.003; subject 2; $\bar{x} = 0.097$, SD = 0.007

Reprinted with permission from Marsolais EB, Edwards BG. Energy costs of walking and standing with functional neuromuscular stimulation and long leg braces. *Arch Phys Med Rehabil*. 1988;69:243-249.

slow and the metabolic efficiency is very low (Table 11-6 and Figure 11-66).[60,61]

An effort is presently made in research centers to overcome the slow walking speed and the high energy consumption of FES by combining this method with some of the commonly used orthoses, including the RGO. In conclusion, because of the low walking speed and the high energy cost of ambulation by electrical stimulation, the use of this method of ambulation is currently limited.

Orthoses for Skeletal Insufficiency

Orthoses for skeletal insufficiency can be used for early mobilization of patients with fractures and to allow conservative management of common joint deformities that result from ligamentous injuries. They should, however, be considered as a supplement or an alternative to a surgical approach.

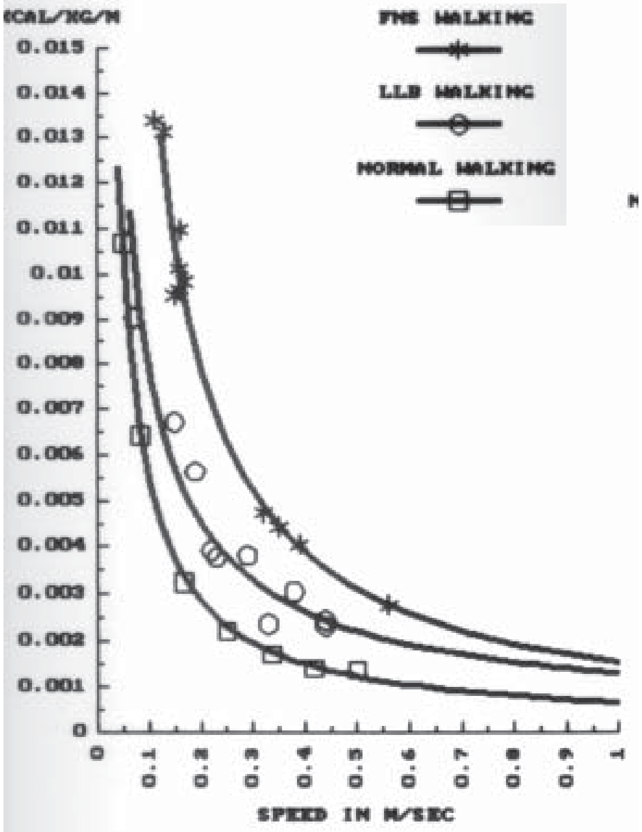


Fig. 11-66. Energy efficiency of paraplegic persons walking with functional nerve stimulation and with long leg braces (LLB) compared with normal walking. Functional nerve stimulation data from Marsolais EB, Edwards BG. *Arch Phys Med Rehabil*. 1988;69:243-249; long leg brace data from the literature; normal subject data from Inman VT, *Human Walking*. Baltimore, Md: Williams & Wilkins; 1981.

These orthoses incorporate two main functional features: (1) reduction of weight bearing on the lower extremity by channeling a major amount of the weight-bearing forces through the ground via the orthosis and, thus, bypassing the skeletal system; and (2) maintenance of bone and joint alignment.

Ischial Weight-Bearing Orthoses

The ischial weight-bearing orthosis uses a cuff design essentially identical to that of the socket for the above-knee amputee (Figure 11-67). The bulge over the femoral triangle pushes the limb backward so the ischium rests on the ischial seat of the orthotic cuff. The plastic cuff is made using lamination techniques or using heat-forming plastics. It can extend all the way to the level of the knee and, therefore, maintain full alignment of the femoral shaft. At the knee, the orthosis usually incorporates a lockable knee joint, as in the KAFO. Below the knee, it may incorporate the equivalent of a PAFO with adequate front closure to preserve proper alignment of tibia and fibula. As an alternative, a metal double-stopped ankle joint can be used in combination with a sole plate to the metatarsal head area. The orthosis is purposely fitted too long, that is, with a clearance of up to one inch between the heel of the foot and the sole of the shoe, to force weight bearing through the orthosis. The transmission of force into the orthosis occurs by way of the ischium into the ischial seat and through the noncompressible soft tissues into the thigh cuff.

Biomechanical studies[62] have compared the effectiveness of the weight-bearing function of this orthosis with different designs, with and without the patient having been trained in their use. The ground reactive force was compared with the amount of force transmitted through the instrumented orthosis. The difference between the two represents the force transmitted through the skeletal system. In general, the weight-bearing function of this orthosis depends on the design and training as follows:

1. The orthosis with a locked knee and a patten bottom produces 100% weight bearing through the orthosis (Figure 11-68).
2. The orthosis with the locked knee, the fixed ankle, and rocker bottom, with training to avoid the pushoff, produces 90% weight bearing through the orthosis. Training consists of teaching the patients to avoid plantar flexion and pushoff within the orthosis, which would load the skeletal system and unload the brace.

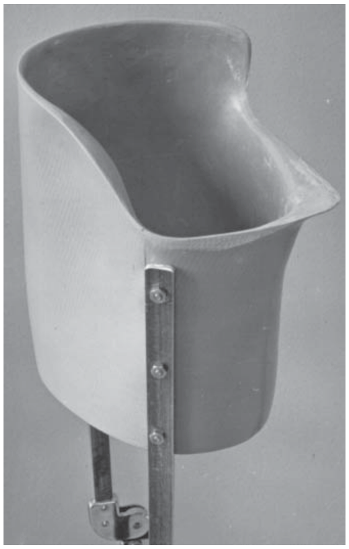


Fig. 11-67. Rigid, quadrilateral cuff for ischial weight-bearing orthoses. Reprinted with permission from Lehmann JF, Warren CG, de Lateur BJ, Simons BC, Kirkpatrick G. Biomechanical evaluation of axial loading in ischial weight-bearing braces of various designs. *Arch Phys Med Rehabil.* 1970;51:331-337.

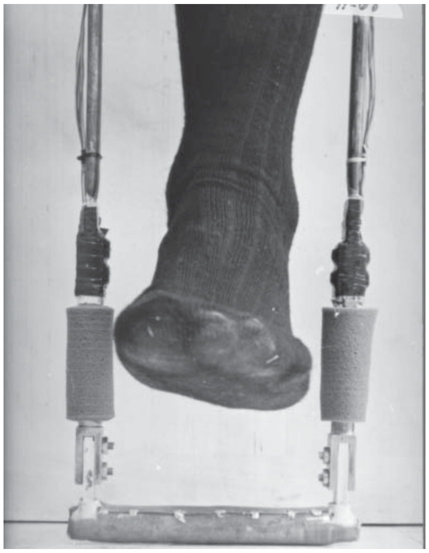


Fig. 11-68. Orthosis with locked knee and patten bottom. Reprinted with permission from Lehmann JF, Warren CG, de Lateur BJ, Simons BC, Kirkpatrick G. Biomechanical evaluation of axial loading in ischial weight-bearing braces of various designs. *Arch Phys Med Rehabil.* 1970;51:331-337.

3. The orthosis with the locked knee and fixed ankle, without a rocker bottom but with training, produces weight bearing through the orthosis at approximately 80% of body weight.
4. The orthosis with the locked knee and fixed ankle, with no training, produces weight bearing through the orthosis at approximately 50% of body weight. Due to the lack of training to avoid pushoff, the patient loads the skeletal system and unloads the orthosis by plantar flexing the foot, pushing against the ground with the forefoot during the latter part of the stance. This reduces the weight bearing from 80% to 50%.
5. The orthosis with the locked knee and free ankle joint, with no training, produces approximately a maximum of 50% weight bearing throughout the orthosis, but only during the heelstrike phase. During pushoff, the lead foot contacts the ground and loads the skeletal system, thereby reducing the weight-bearing function of the orthosis.

In conclusion, the ischial weight-bearing orthosis can reduce weight bearing through the skeletal system (ie, the femoral shaft and below) to variable degrees; the amount of reproduction depends on the modifications of the orthosis and the training of the patient. However, these orthoses cannot be effectively used for protection of the hip joint because only about one third of the transmission of weight occurs at the ischium, which bypasses the hip joint. The rest goes through the hip joint and then through the soft tissues into the cuff of the orthosis.

The weight-bearing orthosis has commonly been used in the form of fracture cast bracing for early mobilization after femoral fractures, and has also been successfully used in cases of delayed union or nonunion fractures. This is presumably because the orthosis maintains the fracture alignment, while at the same time allows some force transmission through the fracture site during functional ambulation, thus stimulating healing.[63,64]

Patellar Tendon Bearing Orthosis

The basic design of the weight-bearing cuff is the same design as that of the patellar tendon bearing socket used for the below knee amputee. For easier donning and doffing, the cuff is halved and the two halves are connected by buckles similar to those of a rigid ski boot. The cuff may be extended to incorporate the ankle joint. The cuff positions the knee into 10° of flexion to allow proper weight bearing through the patellar tendon area, which pushes against the indentation of the cuff. Correspondingly, the fixed ankle joint (Figure 11-69) should be adjusted in 7° of dorsiflexion to accommodate the flexed knee. Again, clearance between the heel and the sole of the shoe can be up to one inch. With a fixed ankle, and training to avoid plantar flexion (pushoff) of the foot, weight bearing can be maximally 50% to 60% of body weight. To avoid weight bearing during pushoff, a rocker bottom may be added.

According to Fry and associates,[65] indications for the short-term use of the orthosis are fractures of the os calcis, postoperative fractures of the ankle, painful conditions of the heel that have been refractory to conservative management, and where surgery is contraindicated. Long-term use of this orthosis has been recommended for delayed union or

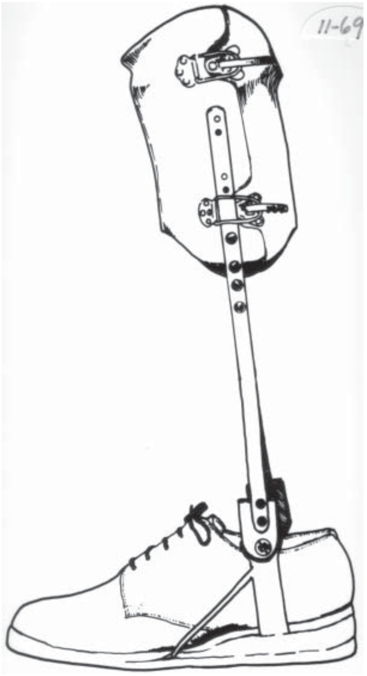


Fig. 11-69. Patellar tendon-bearing brace for limiting weight-bearing, incorporating bivalved patellar tendon-bearing cuff closed by ski boot buckles, standard uprights, double-stopped ankle joint, and sole plate extending to the metatarsal head area. Reprinted with permission from Lehmann JF. Lower limb orthotics. In: Redford JB, ed. *Orthotics Etcetera.* 3rd ed. Baltimore, Md: Williams & Wilkins; 1986.

nonunion of fractures and fusions, avascular necrosis of the talar joint, degenerative arthritis of the talar or ankle joint, osteomyelitis of the os calcis, and sciatic nerve injury with secondary anesthesia that involves the sole of the foot.[65] The only contraindication identified was interference of the orthosis with circulation in the limb. This may be the result of the pressure on the popliteal space which pushes the knee with the patellar tendon area against the front of the cuff, thus interfering with arterial circulation in the limb.

Depending on the design, the functional leg length of any one of the weight-bearing orthoses may be increased. Compensation should be made with shoe lifts on the opposite side.

Orthoses for Control of Knee Alignment

Orthoses can be used to control common knee alignment problems, such as genu recurvatum, or valgus or varus deformity of the knee.

Many knee cage designs are unsuitable for use because they slide up and down on the limb. When this happens, the axis of motion of the cage does not remain aligned with the axis of motion through the anatomical knee joint. As a result, forces are created between the orthoses and the knee, potentially damaging an already injured knee and its ligaments. An effective knee cage (Figure 11-70) consists of a plastic thigh cuff [66] that fits tightly above the femoral condyles and, therefore, prevents downward sliding of the orthosis. Correspondingly, the leg cuff is tightly pulled in below the tibial condyles to prevent upward riding. The two components, upper and lower, are connected by a knee joint that should be properly aligned to approximate the location of the anatomical knee axis of motion and may be polycentric to correct for any slight deviation of an orthotic axis from the instantaneous anatomical knee axis. An extension stop could be added to the joint to prevent hyperextension of the knee. Therefore, this design could be used for correction of genu recurvatum. It also provides a fairly good alignment of the knee and prevents valgus or varus deformities. Complete immobilization of the knee could be achieved if the knee joint is locked. However, this could be achieved less expensively by a cylinder cast or by commercially available braces.

If the cause for the hyperextension of the knee is located at the ankle, a knee cage should not be used to correct a genu recurvatum. Genu recurvatum is often due to the gastrocnemius and soleus contracture with hemiplegia fixing the foot in plantar flexion and, therefore, creating an excessive knee extension moment during the latter part of the stance.[31,32] In this case, physical therapy should be used to stretch out the contracture or, if necessary, heel cord lengthening surgery should be performed. Alternatively, or in addition, a metal double upright AFO, with an adjustable posterior plantar flexion pinstop in combination with a sole plate, should be used. As the patient continues to ambulate, the gastrocnemius and soleus are gradually stretched by adjusting the pinstop so that the foot is fixed more and more in dorsiflexion.

More extensive valgus deformity of the knee, as it is seen in patients with rheumatoid arthritis or other medial collateral ligament destruction, requires the use of a KAFO. The alignment of the knee is maintained by adding a padded dial to the medial upright of the orthosis or extending a plastic cuff from below the knee medially to the knee level. Smith and associates[67] found that a single upright orthosis can be used when the corrective forces do not exceed 18 to 20 lb. With greater forces the double upright orthosis should be used. The maximal knee flexion contracture that can be successfully fitted with this orthosis is 15° to 20°. If any flexion-extension movement is allowed at the knee, it is important that the location of the brace axis coincides as closely as possible with the location of the anatomical axis of the knee joint, or else forces between the

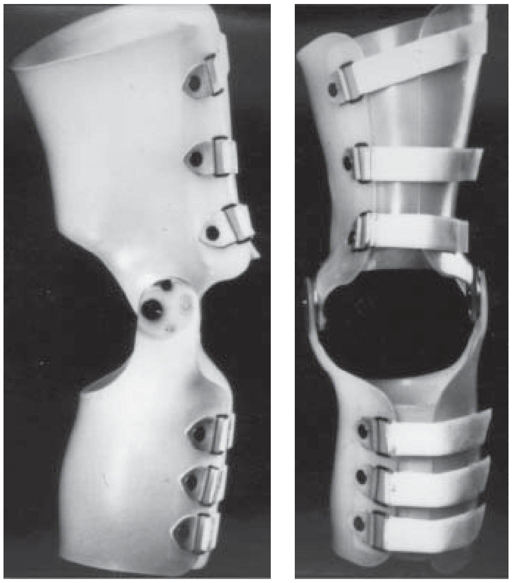


Fig. 11-70. Anterior (left) and medial (right) views of the genucentric knee orthosis. Reprintedwith pemission from Foster R, Milani J. The genucentric knee orthosis—a new concept. *Orthot Prosthet.* 1979;33:31-44.

orthosis and the knee will further injure an already damaged knee joint. Smith and colleagues[67] found that a well-fitted orthosis was well tolerated for periods of 7 to 48 months, thus providing protection against further deformities of the knee and allowing time for healing.

CONCLUSION

In conclusion, the successful application of the orthoses for the upper and lower extremities requires a full understanding of the function of muscles and joints and the biomechanics of orthotic design. Therefore, it is necessary to examine the patient carefully before prescribing a customized orthosis. A "shotgun" approach to orthosis prescription may lead to development of bad habits in the patient, for example, as in prescribing a foot drop orthosis for a stroke patient while disregarding the biomechanical influence of the orthosis on the knee.

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Chapter 12

PREVENTION OF IMMOBILITY COMPLICATIONS THROUGH EARLY REHABILITATION

NICHOLAS T. SPELLMAN, M.D.*

INTRODUCTION

HISTORICAL BACKGROUND

PATHOPHYSIOLOGY

THERAPEUTIC USE OF IMMOBILIZATION

MANAGEMENT OF THE EFFECTS OF INACTVITY ON ORGAN SYSTEMS
  Musculoskeletal System
  Nervous System
  Endocrine and Metabolic System
  Urinary System
  Gastrointestinal System
  Integumentary System
  Respiratory System
  Cardiovascular System

CONCLUSION

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INTRODUCTION

Putting a sick patient to bed has long been a tenet of medical care. Illness and injury require varying degrees of bed rest or immobilization to allow adequate healing. As our understanding of medicine has evolved, immobility itself has been found to be a major contributor in hampering a patient's recovery. In terms of morbidity and mortality, the consequences of immobility are legion. Financially, prolonged immobility is very costly, consuming a large percentage of each healthcare dollar. Militarily, the added morbidity incurred, from contractures and pressure ulcers alone, greatly prolongs the period of convalescence and delays or prevents return to duty. During times of emergency, such delays can potentially interfere with the military medical mission to conserve the fighting strength. Fortunately, awareness, scrupulous nursing care, and early rehabilitation intervention can prevent or minimize most of these complications without placing the patient in jeopardy.

HISTORICAL BACKGROUND

Until the twentieth century, bed rest had been held as the "basic principle in tissue healing."[1] Yet, the clinical importance of exercise as a treatment modality was known in the 16th century. The *Book of Bodily Exercise*, published by Spanish physician Cristobal Mendez[2] in 1533, presented a rationale for the use of exercise in medical treatment. The first modern scientific study to examine the effect of rest on metabolism was published in 1929 by Cuthbertson.[3] The 1940s work of Taylor and colleagues[4] and Widdowson and McCance[5] on the contraction of plasma volume with prolonged bed rest, spurred the current scientific interest in the effects of immobilization. When space travel evolved in the 1960s, the growing interest in the physiologic effects of the antigravity environment further heightened the concerns about immobilization. Hence, there is now a large body of knowledge on the deleterious effects of prolonged immobility.[6-9]

The impact of the consequences of immobilization on the practice of military medicine cannot be underestimated. Delay of return to duty as a result of these complications depletes manpower and undermines readiness. At the time of the Civil War, the strategy of "rest until healed" was in vogue.[10] Regarding casualty management, little changed in subsequent conflicts, undoubtedly contributing to loss of manpower from the insidious iatrogenic complications of immobility. During World War II, an appreciation of the consequences of immobility began to emerge. Building on the early work of Cuthbertson[3] and Dietrick and colleagues[11] observed a reduction in metabolic rate that occurs shortly after immobilization begins.

In 1945, Taylor and colleagues[4] reported reduced blood volume in otherwise normal young males who were put to bed. Widdowson and McCance[5] subsequently confirmed the resultant decrease in plasma volume with concomitant rises in hematocrit and hemoglobin. The deleterious effects of immobility, deconditioning, and contractures, and the importance of early rehabilitation was presented in a report from the Special Exhibit Committee on Physical Medicine in 1946.[12] In the years following World War II, a large body of scientific data regarding the physiologic effects of bed rest emerged. Much of the data were gathered as a result of the rapid development of the space program during the 1960s.[6,7,9]

Hertzman[13] was the first to describe the early rehabilitation of casualties in a combat theater. His reports during the Vietnam War emphasized the importance of proper bed positioning, early ambulation, and early institution of range-of-motion and strengthening exercises. Still, until the Persian Gulf War, no data had emerged to indicate the magnitude of some of the complications of immobility during wartime.

In an analysis[14] of 222 casualties referred to U.S. Army physical medicine services during the Persian Gulf conflict in 1991, 10% had lower limb contractures, 9% had upper limb contractures, 7% had pressure ulcers, and about 38% were referred for strengthening and range-of-motion exercises.[14] These complications adversely impact the length of hospitalization and full functional restoration.

The monetary impact on healthcare in the United States cannot be underestimated. Although no data exist regarding total costs for all the ramifications of immobility, the costs for hospital based pressure sores alone amount to nearly $14 billion per year and an increase of inpatient bed days by as much as 50% in affected patients.[15,16]

PATHOPHYSIOLOGY

Basic to understanding the effects of inactivity on physiologic potential is the definition of functional capacity, maximum physiologic potential, and potential reserve. Kottke[8] defined these terms using the concepts of a person's ability at a given moment in time vs his or her maximum capacity. Functional capacity is the maximum metabolic rate achievable at the casualty's current state of training. Maximum physiologic potential is the maximum metabolic rate achievable in the same subject after a systematic program of physical training. The difference between these two levels is the potential reserve. While the maximum physiologic potential is fixed for a given individual, the functional capacity is dynamic and is a function of the subject's state of fitness. With inactivity, the functional activity falls, yielding an increase in the potential reserve. This gap between functional capacity and maximum physiologic potential must be closed when attempting to rehabilitate a patient who has suffered ill effects from immobility.[8]

Vallbona[17] indicated four types of inactivity that can lead to the complications of immobility:

1. prolonged bed rest,
2. restricted neuromuscular activity,
3. maintenance in a fixed position, and
4. a state of weightlessness.[17]

The first two types are the most important because they are most easily prevented. Maintenance in fixed positions may be unavoidable (such as repair of orthopedic trauma), and weightlessness is of concern only for space travel.

Clinical manifestations secondary to the effects of inactivity can become apparent in a matter of a few days. It is known that with absence of contraction, a muscle will lose up to 3% of its strength per day.[18] The quadriceps and extensors of the back atrophy most rapidly, impairing ambulation and the ability to climb stairs.[19] Bed rest induces decreased oxidative capacity, which can adversely effect endurance.[20] The severity of these complications can be reduced. While at bed rest, strength can be maintained by 20% maximum voluntary isometric contractions of muscle groups for 10 seconds each day.[18] In some medical centers, electrical stimulation has been applied to inhibit atrophy of disuse.[21.22] Gradual progressive tilting of bedbound patients can be utilized as necessary to restore autonomic tone and prevent postural hypotensive episodes prior to resuming ambulation.[7,17]

THERAPEUTIC USE OF IMMOBILIZATION

Although this chapter considers the ill effects of unnecessary immobility, there is no question that immobilization is the proper treatment of many conditions. The most important of these is orthopedic trauma. After the setting of broken bones, restricted motion is required for bone healing postfracture. The immobilization should be restricted to the smallest period required to ensure adequate stabilization, with an eye toward remobilization as early as the treating orthopedist deems safe. Another important example of the proper use of immobilization is the inflamed joint. An acutely inflamed joint, such as in rheumatoid arthritis, should be rested during periods of acute inflammation. In osteoarthritis, there is no inflammatory component, thus there is no contraindication for mobilization.

A long-standing area of controversy is the duration of needed bed rest for a patient who is suffering from uncomplicated acute back pain without neuromotor deficits. There have been many suggested answers to this question, but a recent study[23] has shown that the shorter periods of immobilization result in better outcomes. Objective research[24] suggests that the optimum period of rest should be no longer than 48 hours. Rest beyond this does not produce additional reduction in pain or better clinical outcome, but rather, contributes to further muscle weakening.

MANAGEMENT OF THE EFFECTS OF INACTIVITY ON ORGAN SYSTEMS

Musculoskeletal System

There is considerable loss of strength when a muscle is put at rest. This occurs even with relatively short periods of immobility. Muller[18] has shown that with each day of bed rest there is as much as 3% loss of muscle strength, or up to 20% loss of residual strength per week of immobilization. Lower extremity muscles lose their strength about twice as fast as upper extremity muscles.[25]

This means that there can be as much as a 50% loss of muscle strength in as little as 3 weeks of bed rest. Unfortunately, subsequent recovery of strength proceeds at a much slower pace. Once immobilization has ceased, the most rapid rate of strength recovery is approximately 10% per week.[26] Thus, it is far more efficient to prevent loss of strength secondary to immobilization. Prevention will have important ramifications on the speed at which injured soldiers can be returned to duty.

Ultrastructural Changes in Muscle

Structural changes occur in muscle tissue that has been immobilized. The contractile properties of muscle are adversely effected by immobilization. Davies and associates[27] showed that the mean time to peak tension and the half relaxation time increased significantly with 3 weeks' immobilization of the triceps surae. Maximal voluntary contraction force was reduced. This was associated with a 10% decrease in muscle cross-sectional area. Maximal tension and contraction times recovered within 2 weeks following removal of the long leg cast. Sale and colleagues[28] found that after 5 weeks of immobilization of human thenar muscles there was an average 42% reduction in maximum voluntary contraction force. Likewise, Duchateau and Hainaut[29] found that 6 weeks of immobilization of the adductor pollicis brevis muscle caused a 55% reduction in maximum voluntary contraction force. This correlated with a 19% increase in duration, 15% decrease in amplitude, and 26% decrease in area of the compound muscle action potential recorded from the surface.

Literature regarding fiber type atrophy in immobilization is conflicting. Haggmark et al[30] investigated muscle biopsies taken from the quadriceps of patients immobilized in long leg plaster casts and found a selective slow twitch muscle fiber atrophy with reduced cross-sectional area and reduced oxidative enzyme activity in slow twitch fibers. In his review of muscular atrophy following immobilization, Appell[31] indicated that slow twitch muscles with predominantly oxidative metabolisms are most susceptible to atrophy. The rate of protein synthesis is reduced and catabolism is increased. However, in independent studies, Sirca and Susec-Michieli[32] and Robinson and colleagues[33] found that in patients with osteoarthritis, there was a selective atrophy of fast twitch muscle fibers. This atrophy was interpreted as a direct consequence of decreased muscular activity.

Duchateau and Hainaut[34] showed that the immobilized muscle produced more high threshold motor units than the control muscle. The order of recruitment was maintained, however, in accordance with the size principle of Henneman.[35] The motor unit firing rate at recruitment was unchanged, but maximum firing rate was reduced. This reduction was found to be greatest in low threshold units. Serra and colleagues[36] found that electromyographic (EMG) spectral analysis was altered in patients who sustained muscle atrophy secondary to immobilization, compared to controls. Thus, there is the potential for electrophysiologic evaluation of muscular atrophy secondary to immobilization.

The best strategy to prevent muscular atrophy due to immobilization is to limit the immobilization only to the region of the body in which it is absolutely required, and to make the period of immobilization as brief as possible. In the immobilized patient, an exercise program should still be implemented to retard atrophy. A 30% maximum voluntary isometric contraction (MVC) for 5 seconds each day is sufficient to prevent disuse atrophy.[18] Alternatively, a 50% MVC for 1 second each day will also prevent disuse atrophy.

Vallbona[17] recommends a daily program where the supine patient applies pressure with the feet against a footboard for 5 seconds, relaxing for 10 seconds, and repeating this cycle three additional times.[17] This maneuver requires contraction of most muscle groups of the legs and back. This should not be performed in patients with unstable spines, however. To exercise the upper extremities, a similar routine can be employed by having the patient extend the arms and make a strong grip action. This can be done with or without a hand roll.

The role of electrical stimulation in preventing atrophy from immobilization is controversial, and findings reported in literature are conflicting. Eriksson's[37] group found that intermittent electrical stimulation for up to 5 weeks did not cause any significant changes in enzymatic activities, muscle fiber characteristics, or mitochondrial properties, but did result in improvements in muscle strength comparable to the results of a program of voluntary exercise. More recently, Gibson and colleagues[22] examined the effect of percutaneous electrical stimulation in preventing muscle atrophy secondary to immobilization, by measuring quadriceps mass, composition, and rate of protein synthesis in seven males with tibial fractures who were immobilized in long leg casts for 6 weeks. The results were compared to 14 others with similar injuries who did not receive electrical stimulation. In those not receiving electrical stimulation, quadriceps cross-sectional area (measured by ultrasonography) fell by a mean of 10%, and the rate of protein synthesis was 10% lower than in the control group. In contrast, those who received electrical stimulation showed no significant difference in muscle cross-sectional area or protein synthesis vs the control group.

The impact of electrical stimulation on muscle protein metabolism has also been shown by Bouletreau and colleagues.[38] They demonstrated a reduction in urinary excretion of nitrogen, creatinine, and 3-methyl histidine in intensive care patients who received intermittent muscular electrical stimulation. In a comparison of the effect of isometric exercise vs isometric exercise with electric stimulation on retarding quadriceps atrophy, Arvidsson and colleagues[39] found that there was no difference between the two methods in males, but in females, the results favored electrical stimulation. The degree of atrophy was determined by use of computed tomography. In a study of the effects of electrical stimulation on the quadriceps during postoperative knee immobilization, Morrissey et al[40] found that the decrease in isometric quadriceps torque resulting from immobilization could be signifi-cantly lessened by applying electrical stimulation during the period of immobilization. However, it was found not to significantly alter thigh circumference changes that occurred during immobilization.

Gould and colleagues[41] compared three treatment regimens designed to prevent atrophy of normal thigh and calf muscles immobilized in a long leg cast. One treatment group received nonisometric exercise, the second received isometric exercise, and the third received electrical stimulation. In the electrostimulated group, muscle atrophy was one half that of the exercise group in the thigh and one fifth that of the other two groups in the calf.

In a subsequent study, Gould et al[42] compared isometric quadriceps exercise to electrical stimulation in a group of 20 patients who underwent open meniscectomy. The electrical stimulation applied was a tetanizing, 5-second contraction to the quadriceps approximately 400 times per day. The electrically stimulated group had a significantly reduced loss of quadriceps muscle volume and strength, had reduced postoperative knee swelling, required less pain medication, had a greater range of knee motion, and group members were able to walk earlier without crutches. Buckley and colleagues[21] applied electrical stimulation to major lower extremity muscle groups in four paraplegic patients. Electrical stimulation to achieve tetanic contractions was applied 15 seconds out of each minute for 10 hours per day over 3 weeks. Although the stimulation produced significant growth of thigh and calf muscles, no significant changes in nitrogen balance or phosphate balance could be identified.

Based on these recent studies,[21,41,42] it appears that electrical stimulation is of potential benefit in minimizing immobilization atrophy when immobilization is restricted to a specific anatomic region (such as about the knee). A reasonable protocol is to provide 5 to 15 seconds of tetanizing electrical stimulation to the muscle or muscles of interest per minute for 8 to 10 hours per day. It is not practical to apply electrical stimulation to all major muscle groups of the body for the patient on bed rest, and it is not a substitute for isometric exercise.

Contractures

During immobilization, the balance between collagen synthesis and degradation is altered, resulting in a progressive shortening of connective tissue around joints with contracture formation and loss of range of motion.[43] A muscle immobilized in a shortened position for 1 week will show contraction of the muscle belly with subsequent remodeling of loose connective tissue into dense connective tissue.[19] In addition, immobilization of a muscle in a shortened position can exacerbate immobilization atrophy.[44] If immobilization is necessary, the joint should be maintained in a neutral position in order to keep agonist and antagonist muscles at equal lengths and tensions.[19] The clinical impact of contracture formation results in impaired range of motion, mobility, and ability to perform activities of daily living.

In a study[14] of Persian Gulf War casualties referred for physiatric evaluation, 10% had lower limb contractures compared with 9% in the upper extremity. These contractures could have been due to immobilization for fracture healing, peripheral nerve injuries, or lack of early intervention with range-of-motion exercises. It is unclear whether, or to what extent, these contractures could have been prevented. Haher and colleagues[45] have shown that flexion contractures interfere with the healing of pressure sores, providing further evidence that the complications of immobility are additive and can interact to further hinder recovery.

There are three basic forms of contracture[19(p450)]:

1. soft tissue (skin, subcutaneous tissue, tendons, and ligaments);
2. myogenic (muscle); and
3. arthrogenic (fibrosis and ankylosis of joint capsule).

Soft tissue contracture results from progressive shortening of connective tissue around joints with contracture formation and loss of range of motion.[43] The soft tissue structures include skin, subcutaneous structures, tendons, and ligaments. Myogenic contractures result when a muscle is immobilized in a shortened position with contraction of the muscle belly and subsequent remodeling of loose connective tissue into dense connective tissue[19]; and, as will be discussed below, immobilization of a muscle in a shortened position can exacerbate immobilization atrophy.[44] Arthrogenic contractures result from capsular fibrosis and ankylosis of joints.

Range-of-Motion Therapy

The most important management strategy is preservation of range of motion by moving each joint (either passively, with assistance, or actively) through its full range of motion 3 to 5 times at a minimum of twice a day.[46] The additional steps that can be employed to prevent contractures in the patient at bed rest or one unable to actively move an extremity include proper positioning in the bed.[47]

Simple steps to maintain neutral anatomic position when the patient is in bed and not undergoing range-of-motion therapy should be employed. A firm mattress with solid support is recommended to reduce hip flexion. Starting at the foot, a footboard or boot should be used to maintain ankle position in neutral. Because of gravity and the fact that the posterior compartment of the leg is stronger than the anterior compartment, plantar flexion contractures of the ankle are all too common, particularly if spasticity is present. Trochanteric rolls will assist in preventing external rotation of the hip. Prone lying should be encouraged for at least one half hour per 8-hour shift as an additional measure to prevent hip flexion contractures, as well as to give pressure relief. Periodic side lying, alternating between left and right every 2 hours, will allow for knee and hip flexion.

The superior extremities should be supported with pillows. Positioning of the upper extremities should also alternate at least every 2 hours among three basic positions:

1. shoulder abducted and externally rotated with elbow flexed and forearm pronated,
2. shoulder abducted and internally rotated with elbow flexed and forearm pronated, and
3. shoulder adducted with elbow extended and forearm supinated.

There are two basic wrist and hand positions to provide for alternating flexion and extension. A hand roll can be used to allow flexion of the wrist and hand and, if needed, a wrist hand orthosis can be used to maintain the wrist and hand in extension. Extension and flexion should be alternated at least every 4 hours. It must be remembered that these positioning strategies are adjuncts to, but not substitutes for, an adequate range-of-motion program.

Diathermy

When contracture at a joint is present, use of prolonged stretch within the limits of pain and without tearing of tissues has proven effective, particularly when combined with diathermy.[48-50] As described above, contractures can result from shortening of soft tissue structures, remodeling of muscle tissue, and fibrosis of joint capsules. The effectiveness of a stretching regimen will be enhanced through use of diathermy.

Diathermy is deep heating of biologic tissue. Heating of connective tissue to 41°C to 45°C causes molecular changes in collagen that result in increased extensibility.[51] Superficial heating modalities such as heat packs, whirlpool, and radiant heat lamps do not provide sufficient temperature elevations in muscle and pericapsular structures to effectively alter collagen extensibility.[49,51]

Available deep heating methods include short-wave diathermy, microwave diathermy, and ultrasound diathermy. Of these modalities, ultrasound diathermy is the most commonly utilized and widely available. Ultrasound has the advantage over the other two diathermy methods in that the equipment is much more portable, and its depth of penetration is superior. Both short-wave and microwave diathermy applications rely on conversion of electromagnetic radiation into heat in the tissues and will result in therapeutic temperature elevations at a depth up to 3 cm below the skin surface. Although this is sufficient for adequate warming in superficial muscles and joints, such as the hand, it does not provide for therapeutic temperature elevations in deeper structures, particularly the hip.[52]

Ultrasound utilizes the reverse piezoelectric effect by applying high frequency alternating current to a crystal, thereby producing acoustic vibrations that are converted to heat energy when absorbed by biologic tissues. Ultrasonic energy is selectively converted to heat energy at different tissue interfaces. Bone absorbs approximately 10-fold as much acoustic energy as muscle, which energy is then conducted to the surrounding tissues.[53] For this reason, ultrasound is a very effective modality for achieving therapeutic temperature elevations in contracted joint capsules. Ultrasonic frequencies used in diathermy are on the order of 1 MHz, and ultrasonic intensity is expressed in W/cm^2. To achieve therapeutic temperature elevations in deep tissues, the output of the instrument should be able to attain an average of 4 W/cm^2.[50]

The application of ultrasound diathermy requires the use of a coupling medium or gel to transmit the acoustic energy into the tissue and not reflect it into the surrounding environment. To prevent the occurrence of hot spots and to provide uniform heating, the applicator is moved continuously in multiple planes (stroking technique) about the contracture for 10 to 12 minutes. This should be concurrent with, or immediately followed by, passive stretching to take advantage of the extensibility of the collagen. Dosimetry is determined by patient response to the internal heating, and output should be immediately reduced if the patient complains of pain. Therefore, the patient's nociceptive abilities must be assessed prior to initiating treatment. Contraindications to application of ultrasound diathermy therapy include[51] (*a*) application to anesthetic regions, (*b*) applications over regions of vascular insufficiency, (*c*) application over cemented joint prostheses, (*d*) application over malignant tumors, (*e*) application over the heart, (*f*) application to the eyes, and (*g*) application to the pregnant uterus. When applying it over metallic implants, caution must be taken due to selective heating.

A nonthermal consequence of ultrasound application to biologic tissues is the risk of cavitation. Cavitation refers to the formation and oscillation of gas bubbles within the tissues experiencing ultrasonic energy; these gas bubbles may subsequently coalesce and collapse, resulting in tissue damage. The risk of cavitation is minimized with intensity outputs of under 4 W/cm^2 when a stroking technique is employed. An additional, nonthermal consequence of applying ultrasound is the production of standing waves that can result in blood cell aggregates and stasis of blood flow.[54] Again, this can be avoided if the stroking technique is used.

Splinting

The major goals of splinting are to assist, resist, align, and simulate function.[55] Thus, for sustained stretch or maintenance of neutral position, splinting is useful. The splinting may be static or dynamic. Dynamic splinting has the advantage of allowing the patient to perform active range-of-motion exercises while maintaining the desired positioning when at rest. The splint itself can prevent secondary contracture. Serial casting can be particularly useful in maintaining sustained stretch to reduce contracture.[56] As gains are made, the cast can be reformed to adjust to these gains so that sustained stretch is maintained. By bivalving the cast it can be removed for therapy, exercise, and inspection for areas of excess skin pressure.

Continuous passive motion (CPM) machines have been used for early mobilization of joints in both lower and upper extremities in the early postoperative period.[57,58] Joints are mechanically ranged without patient effort. A predetermined joint range of motion can be applied passively at a slow continuous rate to prevent soft tissue contracture and maintain joint homeostasis and nutrition. During the first few days postoperatively, the CPM machine is applied for up to 12 hours per day. Its use is generally restricted to joints that have been operated upon and it is impractical in many situations because the device is cumbersome. Patient mobility is restricted while the CPM machine is in use.

Whether through manual or mechanical means, early mobilization is the mainstay of contracture prevention. Once formed, contractures are difficult to overcome and require intensive therapy to reverse.

Immobilization Osteoporosis

Bone homeostasis is altered during immobilization. Immobilization in plaster, by bed rest, and the weightless state can all result in detectable demineralization.[59-61] The bony changes of disuse osteoporosis may mimic neoplastic disease, particularly myeloma.[62] Acute osteoporosis has been shown to occur following immobilization and is related to an increase in osteoclastic bone resorption.[63,64] In less than 10 days of immobilization (bed rest), there is an increase in urinary excretion of hydroxyproline, a necessary building block for bone remodeling and maintenance.[65] Similarly, there is up to a four-fold rise in urinary glycosaminoglycans following immobilization.[66] An associated increase in serum calcium and phosphate is observed as well. Serum

1,25-dihydroxyvitamin D decreases significantly during immobilization, reaching its nadir at the end of 1 week. This can lead to a local reduction in bone mass. There is potential for recovery during the early phase of immobilization, but this ability to recover may be lost after 6 months of immobility.[67] Minaire[67] points out in his review of immobilization osteoporosis that the cumulative effect of repeated periods of immobilization in promoting osteoporosis remains hypothetical.

Mechanical stress is an important factor in the maintenance of normal cortical bone remodeling. Immobilization is characterized with reduced osteon densities and cross-sectional area, and with an observed decrease in radiologic density in long bones.[68,69] Vico and colleagues[70] have shown that in rats exposed to 5 days of zero gravity, there is an increase in the number of osteoclasts per square millimeter of trabecular bone.[69] Lips and associates[71] evaluated degrees of mobility and parameters of bone turnover in 70 nursing home residents. An increased urinary excretion of hydroxyproline and a decrease in serum 1,25-dihydroxyvitamin D concentrations were observed, indicating that lower mobility leads to higher bone resorption, which may suppress formation of 1,25-dihydroxyvitamin D.

Bone loss may be averted and potentially reversed by maintaining physical activity.[72] Primary prevention of immobilization osteoporosis is based on exercise, particularly loading of bone intermittently through the day.[67,73,74] However, the type, intensity, duration, and frequency of the exercise has not been determined according to recent literature.[67] Weight bearing should be initiated early during the period of immobilization in order to prevent or possibly reverse osteoporosis.

Drug trials to reverse or prevent osteoporosis secondary to immobilization have shown promise. Administration of 100 International Units (Medical Research Council, London, England) of salmon calcitonin daily, if initiated within the first 2 weeks of immobilization, may retard bone loss and can be effective in preventing or treating immobilization induced hypercalcemia.[67] Recent clinical research[75–79] has centered on antiosteoclastic agents, particularly the bisphosphonates. In a trial to observe the effect of diphosphonate EHDP (disodium ethane-1-hydroxy-1, 1-diphosphonate) on bone mineral metabolism, Lockwood and colleagues[80] evaluated low- and high-dose regimens in four healthy young male subjects placed on bed rest for 20 weeks. At the high dose (20 mg/kg/d) there was some decrease in urinary hydroxyproline excretion and in the rate of bone resorption.

Chappard and associates[64] evaluated the effect of the bisphosphonate (1-hydroxy ethylidene-1,1 bisphosphonic acid) on osteoclast number during prolonged bed rest (120 days) in 15 healthy subjects. Insignificant bone loss and a marked reduction in osteoclast number were observed. Recently, etidronate disodium (disodium diphosphonate) has been found to increase bone mineral density in postmenopausal osteoporosis. Cyclic, low doses of etidronate have been successfully used in multiple studies.[75–77] The regimen is 400 mg/d given over 2 weeks followed by 13 weeks off the medication. This 15-week cycle is then repeated.[78,79] Addition of phosphates provided no additional benefit.[76,79]

Miller and associates[77] compared the effectiveness of cyclic etidronate vs both fluoride and estrogen in postmenopausal osteoporotic women. All three groups had gains in bone mineral density over the 2-year treatment period, but to varying degrees. Sixteen percent gains were observed in the etidronate and fluoride treated groups and a 5% gain was observed in the estrogen treated group. Etidronate, however, was found to be very well tolerated, and superior to fluoride, given the latter's side effect and toxicity profile,[75–77] and was deemed superior to giving supplemental calcium and vitamin D.[77] Calcium supplementation is probably only necessary in postmenopausal patients whose calcium intake is less than 400 mg/d.[81] Whether this regimen will prevent or reverse immobilization osteoporosis remains to be established.

Clodronate, an investigational antiosteoclastic drug, has shown promise in a recent trial comparing its efficacy in preventing demineralization and resorptive hypercalcemia against etidronate and calcitonin.[82] Oral clodronate was found to be superior to etidronate and equally effective as calcitonin.

Currently, there are no satisfactory medications that absolutely prevent immobilization osteoporosis. The best way to prevent this complication is daily weight bearing when possible and stressing bones through exercise.

Nervous System

Nervous system manifestations of immobilization are many. They include sensory, motor, autonomic, central, and psychologic factors. Sensory deprivation in immobilized patients has been associated with altered perception of sensory input.[17,83] This can result in lowered threshold to painful stimuli.

Orthostasis as a result of autonomic nervous system compromise stemming from immobilization is a known complication. This is discussed in more detail in the cardiovascular section that follows. Other autonomic nervous system functions can be adversely effected or exacerbated by immobilization. The course of reflex sympathetic dystrophy, though not occurring directly as a result of immobilization, can be influenced adversely by immobilization. The painful limb involved is not actively moved by the patient due to the sympathetically medicated pain, and subsequently undergoes varying degrees of disuse atrophy and contracture.

Lagier and Van Linthoudt[84] reported two cases of reflex sympathetic dystrophy involving the foot, in which articular changes occurred. The abnormalities included superficial pannus, erosions, and fibrous and bony ankylosis, conditions that can be resolved only through range-of-motion and strengthening programs. Patient tolerance of these programs will determine the ultimate success of rehabilitation. Various physical, medical, and surgical procedures have been employed to alleviate pain and allow ranging and strengthening to prevent and reverse these complications of immobility. These interventions range from contrast baths and desensitization with various textures, to sympatholytic medications and blockades, and surgical sympathectomies. Detailed treatment of reflex sympathetic dystrophy is beyond the scope of this chapter. The reader is referred to the excellent review by Schwartzman and McLellan.[85]

Unlike the autonomic nervous system, the effect of immobilization on the function of the peripheral nervous system is not so clear. Whether an increased risk of peripheral neuropathy results as a consequence of immobility has not been demonstrated in the recent literature. Malathi and Batmanabane[86] demonstrated a reduction in the mean axon diameter in large myelinated fibers of the tibial nerve in cats that was directly proportional to the duration of immobilization. Prolonged immobilization of more than 8 weeks caused splitting of the myelin lamellae and collagen replacement. Marciniak[87] investigated the effect of immobilization on nerve terminals in birds and found reduced numbers of synaptic vesicles and mitochondria. Marciniak reported that prolonged immobilization produced fragmentation of axonal endings with resorption by Schwann cells. Robinson and colleagues[33] demonstrated slower axonal conduction velocities in immobilized limbs of the cat.

Impaired neural control emanating from the central nervous system (CNS) has been described by Haines.[88] Balance and coordination can be adversely affected independently from muscular weakness.[89] Dupui and colleagues[90] found that 30 days of bed rest induced sensorimotor changes that resulted in impaired balance and gait performance. Gait alterations included decreased step length, walking velocity, and stability. Seizures secondary to immobilization hypercalcemia have been reported, and this is discussed in greater detail in the section on the effects of immobilization on the endocrine and metabolic systems.

The association of immobility with dementia has recently been investigated. Selikson and associates[91] examined the risk factors associated with immobility in 34 nonambulatory patients. Factors associated with immobility are contractures, severe dementia, poor vision, and history of fractures. Factors not associated with immobility include age, osteoarthritis, mild to moderate dementia, weight gain, and medications. The effect of immobilization on the development or exacerbation of dementia was not addressed. Intellectual embarrassment, including impaired orientation and concentration, has been shown to occur with immobilization within days.[92]

Psychologic disturbances result as a consequence of immobilization and the attendant reduced sensory stimulation.[83] This may manifest as increased anxiety and be expressed as hostility, worry, jitteriness, tearfulness, insomnia, depressed mood, and feelings of hopelessness.[93] The immobilized patient is at risk for clinical depression and the clinician should be aware of its manifestations, which include the presence of at least five of the following for at least 2 weeks, representing a change from previous functioning[94]:

- depressed mood
- poor appetite or overeating
- insomnia or hypersomnia
- low energy or fatigue
- loss of interest or pleasure
- poor concentration or difficulty making decisions
- feelings of worthlessness
- psychomotor agitation or retardation
- recurrent thoughts of death or suicide ideation.

All of these factors can adversely effect morale and motivation to participate in rehabilitation, and thereby prolong the period of immobilization and reinforce its negative effects.[95-97]

Prevention and management depend on minimizing the period of immobilization and, during that required period, providing adequate sensory stimulation from the environment. Patients should be positioned such that they may interact with their surroundings, particularly with fellow patients and the clinical staff. Patients who have suffered CNS injury, such as right hemisphere stroke, and have resultant left neglect should initially be positioned so that the right side faces the activity of the surroundings as a means of minimizing sensory deprivation. As soon as feasible, patients should be given cognitive tasks, both to maintain mental agility and as a form of interaction and recreation. Examples include puzzles, games, books, and models to build. Emotional support and psychological counseling need to be available to these patients, particularly if the period of immobility is to be prolonged or is associated with concomitant catastrophic illness.

Endocrine and Metabolic Systems

There are multiple endocrine and metabolic derangements that occur as a result of immobility.[56] Metabolic rate becomes reduced within days of immobilization and may persist for several weeks after remobilization.[6,11,98] Electrolyte homeostasis is altered in immobility such that there are increased losses of calcium, sodium, potassium, phosphorus, sulfur, and nitrogen.[98,99] The negative nitrogen balance reflects a state of increased protein catabolism that overtakes protein synthesis.[98]

Although profound hypercalcemia is rarely associated with immobilization, elevated parathyroid hormone levels have occurred in association with immobilization.[100] Henke and colleagues[101] reported a case of a 13-year-old boy who developed hypercalcemia 1 month after a femoral fracture and presented with progressive anorexia, nausea, vomiting, irritability, and respiratory arrest as a result. Weissman and colleagues[102] reported a case of unexplained intermittent hypercalcemia in a critically ill patient who was thought to be influenced by immobilization. Although the issue of immobilization was not specifically addressed, Forster and associates[103] reported a 15% incidence of hypercalcemia in 100 critically ill patients who had a longer than average length of stay (greater than 12 days) in the intensive care unit (ICU).

The mechanism of immobilization-induced hypercalcemia is a result of enhanced regional blood flow in the bone and increased osteoclastic activity.[63] Vico and colleagues[70] showed that in rats exposed to weightlessness for as little as 5 days, the number of osteoclasts per square millimeter in trabecular bone is significantly increased. Suppression of the increased osteoclastic activity with the resultant increased bone turnover and hypercalcemia is the rationale for treatment with calcitonin.

Treatment of severe hypercalcemia (> 13.0 mg/dL) consists of restricting dietary calcium and medications that might cause hypercalcemia (such as vitamin D and thiazide diuretics), administering intravenous isotonic saline (2.5 to 4.0 L/d) with simultaneous diuresis with furosemide or ethacrynic acid, and supplementing phosphate.[104] Mithramycin is sometimes used as well, especially for hypercalcemia associated with malignancy. Treatment with calcitonin, particularly when used in combination with glucocorticoids, may be of benefit, as both agents are inhibitors of bone resorption; calcitonin also enhances renal excretion of sodium.[105,106] Usual doses are 4 to 8 units per kilogram of body weight subcutaneously or intramuscularly every 6 to 12 hours. A major disadvantage of calcitonin is that it cannot be taken orally. Adverse effects may include mild nausea, abdominal cramps, flushing, and more rarely, an anaphylactic reaction.

Bisphosphonates, such as etidronate, bind to hydroxyapatite in bone and prevent dissolution of bone. They also inhibit osteoclast function. Etidronate and pamidronate have been approved for use in the United States.[107] Although available in oral preparation, their absorption from the gut is poor, so intravenous administration is preferable in acute hypercalcemia. The recommended dose of etidronate is 7.5 mg/kg of body weight intravenously over 4 hours daily for up to 7 days. Adverse reactions are fortunately mild and include altered taste perception, allergic reaction, transient temperature elevation, transient leukopenia, and slight serum phosphate reductions.[107]

Clodronate, an investigational bisphosphonate antiosteoclastic drug, has shown promise in a recent trial[82] comparing its efficacy in preventing demineralization and resorptive hypercalcemia to that of etidronate and calcitonin. Oral clodronate was found to be superior to etidronate and equally effective as calcitonin. The mineralization defects observed during prolonged treatment with etidronate were not observed with clodronate.

Potassium and magnesium depletion are to be expected as a consequence of therapy, so close monitoring of electrolytes, with replacement as needed, should be undertaken. The clinician should have a high index of suspicion for electrolyte perturbation in any immobilized patient who develops anorexia, nausea, vomiting, irritability, and mental status changes.

Insulin production remains normal during immobilization but glucose intolerance has been reported.[108] This is believed to occur as a result of increased peripheral insulin resistance.[109] Further effects of immobilization include increased serum cholesterol levels with decreased low density lipoprotein levels.[56] Other reported endocrine effects of immobilization have been amenorrhea, and low levels of gonadotropin and estrogen.[63]

Urinary System

Urinary system management during immobilization is an often neglected part of patient care and usually only comes to attention when the patient becomes incontinent or oliguric. The major preventable complications are dehydration, renal lithiasis, and urinary tract infection.

Adequate fluid intake is simply assessed through use of intake and output monitoring as well as body weight measurements, skin turgor, and mucous membrane moisture. Unless there is a medical indication to restrict fluid intake, minimum water requirements for an adult are on the order of 2 to 2.5 L/d.[110] (This assumes no contraindications to free water administration such as renal failure, congestive heart failure, and syndrome of secretion of inappropriate antidiuretic hormone [SIADH], to name a few.)

Laboratory studies can also assist with assessment of hydration status. These include urine specific gravity, blood urea nitrogen (BUN), creatinine, and electrolytes (particularly sodium). Elevation of the BUN, especially in the face of a stable creatinine and rising urine specific gravity, and elevation of serum sodium can herald dehydration. If the patient cannot take free water orally, intravenous supplementation will be necessary. Maintenance of adequate fluid intake is of particular importance in the early stages of immobilization. Recumbent positioning initially increases the circulatory blood volume subsequent to resorption of extravascular fluid. The ensuing diuresis is accompanied by urinary excretion of sodium, potassium, calcium, and phosphorus.[9]

Hypercalcuria, particularly in the presence of urinary stasis, places the patient at risk of upper and lower urinary tract calculi. Up to 30% of immobilized patients develop lithiasis (Figure 12-1).[111] It is felt that bed rest itself increases the risk for the formation of calcium-containing renal stones. Hwang and associates[112] have shown that after 5 weeks of bed rest, the mean urinary calcium excretion rose during the first week of the 5 weeks of bed rest by 32% and remained elevated. Mean urinary phosphorus excretion increased by more than a factor of 10. Urinary excretion of sodium, urate, and magnesium rose slightly. Urinary pH, oxalate, and citrate changed very little. Urinary concentrations of calcium phosphate, calcium oxalate, and monosodium urate increased during the period of bed rest, showing that the propensity for crystallization of stone-forming salts is enhanced with bed rest. In fact, struvite and carbonate-apatite bladder calculi are most commonly encountered.[56] However, in the absence of hypercalcemia, it is not necessary to reduce dietary calcium intake.[113]

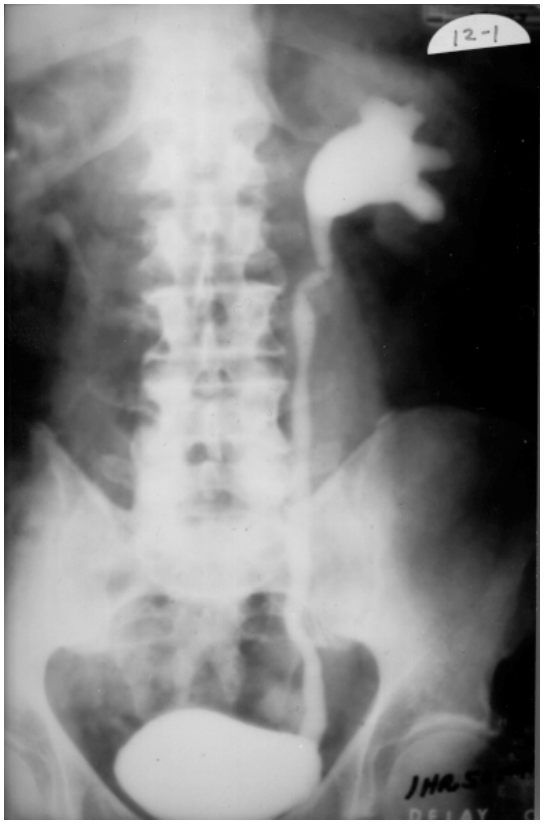


Fig. 12-1. Intravenous pyelogram showing left proximal urinary tract obstruction with hydronephrosis secondary to calculus. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

There is increased risk of urinary tract infection in immobilized patients, particularly when there is stasis of urine. Colonization in and of itself is not justification for treatment with antibiotics, as this merely increases the risk of producing antibiotic resistant organisms. The exception to this is urea-splitting bacteria such as *Proteus* species. Because of the increased risk of struvite stone formation, colonization with urea-splitting organisms should be eradicated.[114,115] Otherwise, antibiotic treatment should be reserved for clinically apparent infection by observing white blood cells (WBCs) in the urinalysis (> 8 WBC per milliliter of midstream urine or > 20 WBC per high powered field of centrifuged urine) associated with proteinuria, bacteriuria and clinical signs of infection.[116,117] In this manner, if and when a urinary tract infection develops, the treated organism will be minimally resistant. Prophylactic measures include adequate urinary flow and frequent, complete drainage of urine from the bladder. If, however, the patient has a history of more than two urinary infections per year, prophylaxis with nitrofurantoin or trimethoprim-sulfamethoxazole may be prudent both from clinical and cost-effectiveness standpoints.[118]

It must be ascertained that the immobilized patient is voiding the bladder completely to minimize the risk of urinary tract infection and reflux of urine into the upper urinary tract. This is best confirmed by determining the postvoid residual volume, which involves placing a catheter into the bladder immediately after the patient has voided. Normally, the residual volume should be less than 75 mL. If it is more than 75 mL, there is incomplete emptying.[117,119] To ensure complete emptying of the bladder, a program of intermittent catheterization is recommended.

The frequency of catheterization in the continent patient is dependent on the volume of the postvoid residual. If the postvoid residual volume is between 75 and 150 mL, the patient should be catheterized once daily; if it is between 150 and 250 mL, the patient should be catheterized twice daily; if it is between 250 and 350 mL, the patient should be catheterized three times daily; and if it is between 350 and 450 mL, the patient should be catheterized four times daily. Postvoid residual volumes greater than 450 mL require more frequent catheterizations, up to every 4 hours.[117,119] Intermittent catheterizations more frequently than every 4 hours are not practical from a patient care standpoint and will not allow the patient adequate sleep. Also, the primary advantage of intermittent catheterization over an indwelling catheter is the reduced risk of urinary tract infection. More frequent intermittent catheterization will negate this advantage, and thus, indwelling catheterization may be preferable. Indwelling catheters are also preferable when urine volumes are fluctuating considerably due to changes in the patient's volume status.

The various etiologies of retained urine are beyond the scope of this chapter but may involve outlet obstruction, lower motor neuron disease, upper motor neuron disease, or intrinsic damage to the bladder wall. Differentiation of these etiologies requires urodynamic study. Fluoroscopy is recommended during the urodynamic study to determine if reflux of urine into the upper tract is present. If reflux is present, the risk of hydronephrosis and pyelonephritis is a concern.

In patients with retention of urine who are undergoing intermittent catheterization, use of urinary antiseptics has been advocated as a means of reducing the likelihood of infection.[120] However, the use of prophylactic antibiotics remains controversial.[121,122] Clinical advantage in instituting antibiotic treatment for prevention of urinary infections has not been conclusively demonstrated, and recent studies tend to indicate that prophylactic antibiotics do not have a long term effect in reducing bacteriuria.[121,122]

Gastrointestinal System

Nutrition

One of the often overlooked risks of immobilization is its effect on the gastrointestinal system. As indicated above, metabolic abnormalities caused by immobilization, particularly immobilization hypercalcemia, can present with gastrointestinal complaints including anorexia, nausea, and vomiting.[101] The anorexia associated with illness also places the bedridden patient at increased risk for malnutrition. Vigilance in the form of calorie counts; frequent weighing of the patient; and monitoring of serum albumin, protein, hematocrit, hemoglobin, and WBC count can serve as markers to evaluate the patient's nutritional status. If the patient is unable to maintain adequate intake, nutritional supplements in the form of enteral or parenteral support will be required to maintain adequate nutrition.[123] It must be remembered that during illness, the metabolic rate is increased, and without adequate nutrition, excessive catabolism will occur. And, as will be seen in the Integumentary System section, inadequate nutrition and increased catabolism are particular hindrances in the prevention of pressure sores.

Constipation

The most commonly encountered gastrointestinal complication of immobilization is constipation. Kinnunen[124] studied 439 patients and found the relative risk for constipation at 1.7 in patients who walk less than 0.5 km daily. The relative risk for bedbound patients is nearly 16 times that for a person who walks more than 0.5 km per day. The prevalence of constipation was directly correlated with fecal incontinence. Decreased peristalsis secondary to increased adrenergic tone may play a role in the development of constipation in the immobilized patient. Decreased fluid and dietary fiber intake, which is common during illness, will also contribute to the development of constipation. Therefore, the best way to avoid this complication is to mobilize the patient. If this is not possible, an adequate bowel program is required.

The goals of a bowel program are to ensure regular and complete evacuation. Initially, this should be a daily routine at a specified time. To make optimum use of the gastrocolic reflex, the program should follow a meal by 30 to 60 minutes. Usually, this is following breakfast or the evening meal to minimize disruption of the daily diagnostic or rehabilitation program, or both. Once regularity is established, the frequency of the program can be reduced to an optimal evacuation pattern of three times per week. If the patient is incontinent of stool, the possibility of fecal impaction should be investigated, as this can hinder the establishment of a successful program. Impaction should especially be considered if the patient has been treated with medications that have a constipating effect, such as narcotics. If this is the case, disimpaction, treatment with laxatives, enemas, or any combination will be necessary to clear the lower gastrointestinal tract prior to initiating the bowel program. Objectives of the program are to provide adequate bulk and softness to the stools such that laxatives can generally be avoided.

Regular defecation can be stimulated reflexively through distention of the rectum. This can be achieved through use of a glycerin suppository or by digital stimulation by placing gloved digits into the rectum and sweeping in a circular pattern at the time desired for defecation. Adequate intake of dietary fiber greatly enhances the effectiveness of a bowel program by providing adequate stool bulk. Recommended dietary soluble fiber intake should exceed 10 g/d. If this is not possible from the diet, supplemental fiber should be prescribed. One such regimen is to consume 3 to 4 grams of psyllium hydrophilic mucilloid fiber (Metamucil [Procter and Gamble; Cincinati, Ohio]) in 250 mL of water three times per day. This will also help to maintain adequate free water intake to promote soft stools. (This assumes no contraindications to free water administration such as renal failure, congestive heart failure, or SIADH, to name a few.) Psyllium is contraindicated if fecal impaction or intestinal obstruction is present.

If softening of the stool is necessary, this can be accomplished through use of a nonabsorbable softening agent, such as docusate sodium (Colace [Mead Johnson Pharmaceuticals; Evansville, Indiana]) 100 mg by mouth twice a day. Digital stimulation or a glycerin suppository is then used to initiate defecation as desired. The recommended initial bowel program is as follows: (*a*) rule out impaction, (*b*) maintain adequate hydration (> 2 liters free water orally per day), (*c*) administer psyllium hydrophilic mucilloid (3 to 4 grams 3 times daily), (*d*) administer docusate sodium (100 grams 2 times daily), and (*e*) use a postprandial glycerin suppository or digital stimulation once daily. The dosage of soluble fiber and stool softener will likely need to be titrated to optimize consistency and to ease completeness of defecation; but the primary goal remains regular, controlled defecation.

Peptic Ulcer Disease

Patients facing physiological stress have long been known to be at risk for peptic ulcer disease.[125-127] Lev and colleagues[128] examined increased stress ulcer occurrence in the battle wounded during the Vietnam War. It has also been shown experimentally that immobility is a risk factor for ulceration.[129,130] Clinically, the incidence of stress ulcers due to immobility alone is not known, but in spinal cord injured patients who are physiologically stressed and presumably immobilized, the incidence of stress ulcers has been reported to be 4%.[131,132] In Kewalramani's[131] study, 42% of patients with gastric or duodenal ulceration developed perforation. Kuric et al[132] and Dietz et al[133] have indicated that adequate nutrition (meeting the patient's total energy requirements) will reduce the incidence of gastric ulceration by more than half. Anorexia and nausea are early symptoms.[134] Melena is the usual clinical presentation.[135]

The diagnosis is made by positive barium contrast upper gastrointestinal tract radiography or fiber optic endoscopy, or both (Figure 12-2). Of those patients with gastrointestinal bleeding, in the spinal cord injured population, 63% had duodenal ulcers; 21% had gastric ulcers; and approximately 5% each had gastritis, esophagitis, Mallory-Weiss tear, or gastric carcinoma.[135] Use of anticoagulants and steroids can further increase the risk of gastrointestinal bleeding.[136] In the spinal injured population, administration of decadron in the first 48 hours postinjury has not been shown to be associ-

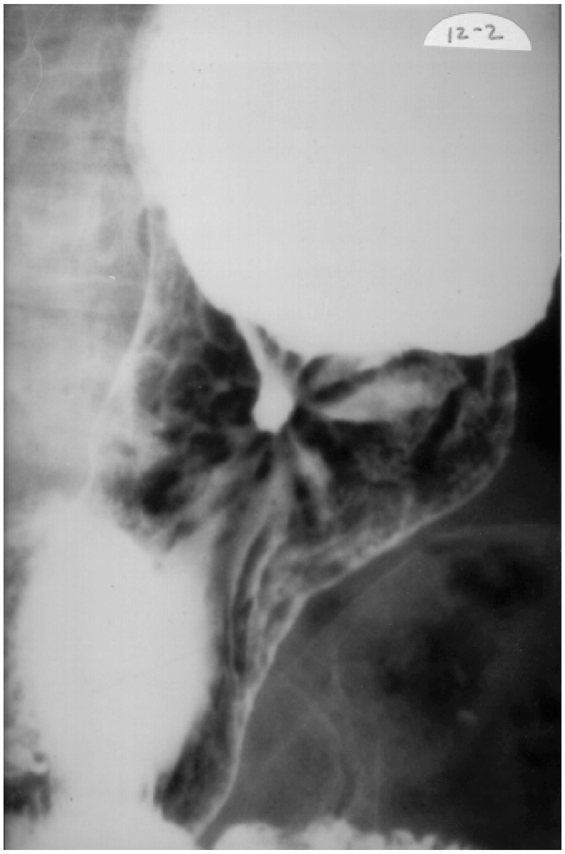


Fig. 12-2. Upper gastrointestinal series demonstrating an acute gastric ulcer. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

ated with ulcers, but it has been associated with pancreatitis.[137]

As indicated above, the important preventive strategy for stress ulcers is adequate nutrition.[132,133] Additional suggested medical measures are administration of antacids, (H_2) Histamine$_2$-receptor antagonists, sucralfate (Carafate [Marion Merrill Dow; Kansas City, Missouri]), Omeprazole (Prilosec [Merck Sharp and Dohme; West Point, Pennsylvania]), and misoprostol (Cytotec [G.D. Searle and Co; Chicago, Illinois]).[138-142] H_2-receptor antagonists remain the mainstay of prevention and treatment. H_2-receptor antagonists inhibit the action of histamine at its receptor on the gastric parietal cell, resulting in decreased acid secretion.[143,144]

Antacids (magnesium hydroxide and aluminum hydroxide combinations) are comparable to H_2-receptor blockers in treatment of duodenal ulcers, but their use in prevention of gastric and duodenal ulcers and for treatment of gastric ulcers is not well established.[145] Their primary method of action is acid neutralization. Although sucralfate has been shown to be as effective as H_2-receptor blockers in the treatment of duodenal ulcers, it has not been approved by the Food and Drug Administration for treatment of gastric ulcers.[146] Its exact mechanism of action is not known, but it appears to have cytoprotective effects.[141] Omeprazole is a proton pump inhibitor that reduces the secretion of hydrogen ions from the parietal cell. MacLellan and colleagues[140] have shown that in spinal cord injured rats, omeprazole is more effective than antacids or H_2-blockers in preventing ulceration. In humans, omeprazole heals ulcers more rapidly and with fewer failures than H_2-receptor antagonists, but it is very expensive. It is best reserved for the treatment of refractory ulcers; the suggested regimen is 40 mg/d for 8 weeks.[147-149]

Prostaglandin analogues, such as misoprostol, inhibit acid secretion from the parietal cell and stimulate bicarbonate and mucus secretion from the gastric mucosa. In terms of healing peptic ulcers, misoprostol is less effective than H_2-receptor antagonists.[150] Misoprostol is indicated for the prevention of gastric ulcers for patients who are on chronic nonsteroidal antiinflammatory drugs.[151-154] Fabian and colleagues[142] completed a study of 278 ICU patients that indicates that both an H_2-receptor antagonist (cimetidine) and sucralfate are effective for stress ulcer prophylaxis in severely injured patients. A suggested regimen is presented in Table 12-1. With the exception of

TABLE 12-1

SUGGESTED ORAL REGIMEN FOR THE PREVENTION AND TREATMENT OF PEPTIC ULCERS

Medication	Dose/Frequency
H_2-receptor antagonists	
Cimetidine	800 mg once daily
Famotidine	40 mg once daily
Nizatidine	300 mg once daily
Ranitidine	300 mg once daily
Sucralfate	1 g four times daily
Anatacid	10 cm^3 four times daily
Omeprazole	40 mg once daily x 8 wk for refractory ulcer
Misoprostol	200 µg four times daily if patient is taking NSAIDs

NSAID: nonsteroidal antiinflammatory drug antacids, all the medications listed in Table 12-1 should generally be avoided in pregnancy due to the lack of controlled data on their teratogenic effects on the human fetus. Misoprostol is contraindicated in pregnancy due to its abortifacient property.

Superior Mesenteric Artery Syndrome

A rare, but potentially life threatening, gastrointestinal complication concomitant with immobility is the superior mesenteric artery syndrome. This syndrome results from a loss of mesenteric fat coupled with bed rest (supine immobilization), which causes the superior mesenteric artery to obstruct the upper gastrointestinal tract by compressing the third part of the duodenum against the underlying aorta.[155] It tends to be more common in females than males, in younger ages (14 to 19 years), and in asthenic body habitus.[156]

The clinical presentation includes nausea and voluminous, bilious postprandial projectile vomiting. There may be periods of normal appetite and the presence of bowel sounds if the obstruction is intermittent.[156] Diagnosis is confirmed with an upper gastrointestinal cinefluoroscopy (Figure 12-3). The primary interventions are nasogastric drainage with intravenous hydration and nutrition. Eating in the left lateral decubitus or upright positions may help to alleviate symptoms. In the Hutchinson and Bassett report[156] on 14 patients, all responded to conservative management, with none requiring surgical decompression; although 50% had more than one episode that required treatment.

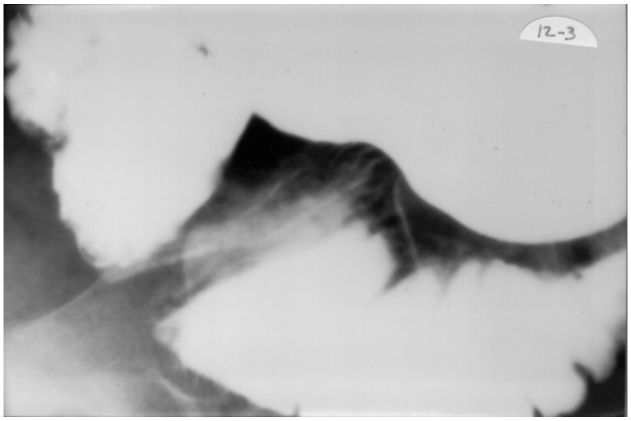


Fig. 12-3. Upper gastrointestinal series demonstrating the superior messenteric artery syndrome with partial obstruction of the inferior duodenum. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

Less commonly, other potential gastrointestinal system emergencies that can complicate the recovery of the immobilized patient include pancreatitis and small-bowel obstruction, or ileus. Awareness is the primary means of prevention for all of the gastrointestinal complications of immobility.

Integumentary System

Pressure ulcers are among the most costly complications of immobility in terms of added morbidity and cost. Prevalence in the acute care setting is estimated to be between 3% and 28%; with an incidence of 1% to 8% during hospitalization.[157] In spinal cord injured patients, development of a pressure ulcer is associated with a doubling of inpatient hospital days and an increased cost of up to $40,000 per occurrence.[158] Cost estimates in the non–spinal-injured population during acute hospitalization are nearly $4,000 per occurrence.[159] Not surprisingly, the associated costs are lower in the chronic care setting.[160] Therefore, the prevention of pressure ulcers is of great importance in minimizing the morbidity of immobilization and in the conservation of healthcare dollars.[161,162] Up to $14 billion can be saved if pressure ulcers are prevented.[16]

Pressure ulcers are localized areas of cellular necrosis due primarily to increased pressure over a focal area of soft tissue for a sufficient length of time to result in ischemia.[163] Sustained pressures (as low as 32 mm Hg), shear forces, friction, moisture, increased skin temperature, hypoproteinemia, and anemia have all been found to be important in the pathogenesis of pressure ulcers.[164-172] Tissue subjected to pressures of more than 32 mm Hg for extended periods of time will cause capillaries to collapse, resulting in tissue ischemia, thrombosis, cell death, and ulceration.[172] Pressures of 70 mm Hg applied to the skin for only 2 hours can result in irreversible tissue injury.[173]

Pressure distributions over healthy skin is dependent on the patient's position in bed. Figure 12-4 illustrates the varying pressure gradients in the supine and prone positions.[174] The areas exhibiting the highest pressures are at the greatest risk for ulceration. In the supine position, pressures sufficient enough to cause tissue ischemia are found over the occiput, spine, sacrum, and heels. In the sitting position, pressure over the ischial tuberosities can approach 300 mm Hg.[163,166,174] Patients with insensate skin, impaired mental status, or inability to perform pressure relief are at even greater risk for development of pressure ulcers.[172,175] These ulcers can develop over any bony prominence but are most com-

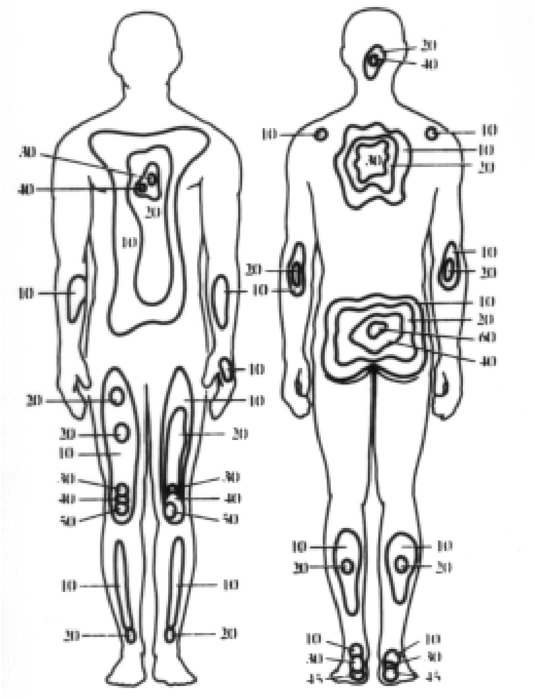


Fig. 12-4. Pressure distributions in recumbent healthy adult male (mm Hg). Left: Prone. Right: Supine. Adapted with permission from Lindan O, Greenway RM, Piazza JM. Pressure distribution on the surface of the human body: 1. Evaluation in lying and sitting position using a "bed of springs and nails." *Arch Phys Med Rehabil*. 1965;46:378.

TABLE 12-2

SITES OF PRESSURE SORES IN PARAPLEGIC PATIENTS

Site	Percentage
Ischial tuberosity	28
Trochanter	19
Sacrum	17
Heel	9
Malleolus	5
Pretibial	5
Patella	4
Foot	3
Anterosuperior spine	3
Elbow	2
Other (including occiput and costal margin)	6

Source: Dansereau JG, Conway H. Closure of decubiti in paraplegics. *Plast Reconstr Surg*. 1964;33:474-480.

mon over the ischial tuberosity, greater trochanter, sacrum, heel, malleolus, and tibia (Table 12-2).[176]

In Persian Gulf War casualties, the prevalence of lower extremity pressure ulcers was 3%.[14] This compares favorably to the reported incidences of up to 8%.[157] Upper extremity and torso ulcers were even less frequent, at 2% each. Still, pressure ulcers are completely avoidable, and the mainstay of management is prevention.

Several tools have been developed to assist in identification of patients at risk.[177-179] An example of an instrument for identification of patients at risk for pressure sores is presented in Exhibit 12-1.[177]

EXHIBIT 12-1

INSTRUMENT FOR IDENTIFICATION OF PATIENTS AT RISK FOR PRESSURE SORES

A. Mental Status
  5 Alert
  4 Apathetic
  3 Confused
  2 Stuporous
  1 Unconscious
B. Continence
  4 Fully controlled
  3 Usually continent
  2 Minimally continent
  1 Uncontrolled
C. Bed Mobility
  4 Full
  3 Slightly limited (requires minimal assistance)
  2 Very limited (requires moderate assistance)
  1 Immobile
D. Activity
  4 Ambulatory without assistance
  3 Walks with assistance
  2 Chairfast
  1 Bedfast
E. Nutrition
  3 Good: eats a balanced diet
  2 Fair: occasionally refuses meal
  1 Poor: seldom eats complete meal/dehydrated

Maximum score = 20; Minimum score = 5. High risk of pressure sore if score ≤ 11. Reprinted with permission from Gosnell DJ. An Assessment Tool to Identify Pressure Sores. *Nurs Res*. 1973;22:55-59.

Normally, a person changes bed position as frequently as four times an hour, and, ideally, patients should relieve areas of pressure for 5 seconds every 15 minutes, although this may be impractical.[180] For bedridden patients, a practical minimum is to turn them at least once every 2 hours.[181,182] For the patient using a wheelchair, pressure relief (particularly over the ischial tuberosities, sacrum, and trochanters) should be initiated at least every 30 minutes for a duration of 15 seconds.[183] Proper positioning of the patient at bed rest will help minimize the development of pressure ulcers and will assist with contracture prevention.[15,47,184] The basic positioning strategies are described in the section on contractures.

Use of pressure relieving devices such as seat cushions, special beds, mattresses, and bolsters can be of assistance in redistributing pressure.[185] Maklebust and associates[186,187] found that the air-fluidized bed (Clinitron [Hill-Rom Company, Inc.]), low-air-loss bed (Flexicare [Hill-Rom Company, Inc.]), and three-layered air cushion (Sof-Care [Gaymer Industries, Inc.]) all reduced tissue interface pressures below 32 mm Hg. Use of therapeutic air suspension beds has been shown to reduce the incidence of pressure ulcers in the intensive care unit setting by 79%.[16] Several of these devices are illustrated in Figures 12-5, 12-6, and 12-7. However, none of these measures affords complete pressure relief, particularly beneath the heels, and are not substitutes for frequent skin checks and regular pressure releases.[186] Inspection of susceptible areas (see Figure 12-4 and Table 12-2), meticulous attention to skin hygiene, and adequate nutrition are of utmost importance.[15,172] Maintenance of adequate hydration and nutrition is essential both for prevention of ulceration and for healing, should a pressure sore develop. Increased tissue pressure and shear, combined with skin atrophy from poor nutrition, accelerate the development of decubiti.

Although preventable, pressure ulcers do occur. The classification of pressure ulcers is dependent

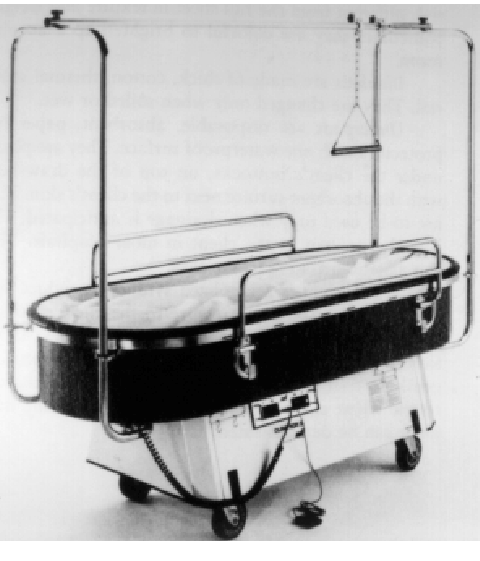


Fig. 12-5. Clinitron air-fluidized therapy bed. Designed to relieve pressure in the recumbent patient. Except at the heels, reduces tissue interface pressures below 32 mm Hg.[185,186] Reprinted with permission from Hill-Rom Company, Inc., Batesville, Indiana.

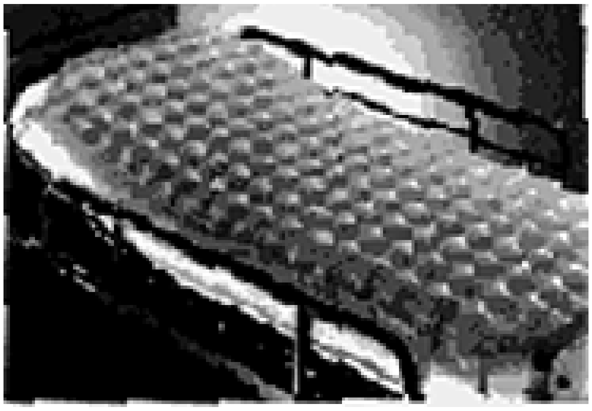


Fig. 12-6. Sof-Care alternating pressure mattress. Designed to relieve pressure in the recumbent patient. Except at the heels, reduces tissue interface pressures below 32 mm Hg.[185,186] Reprinted with permission from Gaymar Industries, Inc., Orchard Park, New York.

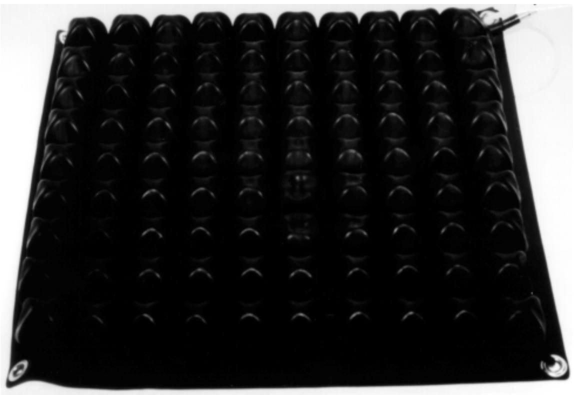


Fig. 12-7. ROHO low profile dry flotation cushion. Designed to relieve pressure in the sitting patient. Reprinted with permission from ROHO Incorporated, Belleville, Illinois.

TABLE 12-3

GRADING OF PRESSURE SORES

Classification	Description
Grade 1	Skin erythema that persists > 24 h and does not blanch to digital pressure
Grade 2	Ulceration of dermis
Grade 3	Ulceration into subcutaneous fat
Grade 4	Ulceration of muscle
Grade 5	Ulceration into body cavity (including bursae and bone)

Adapted with permission from Daniel RK, Hall EJ, MacLeod MK. Pressure sores-a reappraisal. *Ann Plast Surg*. 1979;3(1):53-63.

on the depth of tissue injury.[188] The grading of pressure sores is summarized in Table 12-3. In the earlier stages of ulceration (grades 1 and 2), conservative management can be effective.[189] Blanchable erythematous skin should return to normal in 1 day if pressure is relieved. Nonblanchable erythematous skin should heal in 1 to 3 weeks if corrective action is taken. Bullae may require 1 month or more to heal.[172] It is therefore important that the developing decubitus ulcer be addressed. The most obvious reason is that the more superficial the ulcer, the less morbidity and cost will be encountered. The risk of bacterial superinfection will be minimized as well.

Malignant degeneration has also been reported to occur in long-standing ulcers.[190] As with ulcer prevention, relief of pressure and friction over the injured area is the primary treatment. Nutrition and hydration should be optimized to hasten wound healing. Necrotic tissue should be debrided. Frantz and colleagues[191] have conducted a 5-year retrospective trial that revealed over 70 different treatments used to treat pressure ulcers. Approximately 40% involved open-air or dry gauze treatments and in 60%, an antiseptic solution was used. A subsequent clinical trial has indicated that moist dressings applied three to four times daily are the conservative treatment of choice.[191]

Wounds of grades 3 through 5 will usually require surgical attention. Surgical management includes skin grafts, skin flaps, muscle flaps, musculocutaneous flaps, and free flaps in progressing order from superficial to extensive treatment. The reader is referred to surgical literature for a detailed discussion of these options.[192] Postoperative complications to be wary of include hematoma, seroma, and wound infection. It should be noted that recurrence of ulceration in the same region is as high as 67%.[193] The roles of patient education and prevention along with meticulous postoperative care cannot be overstated.

Respiratory System

A major adverse effect of immobilization on the respiratory system is of a restrictive nature.[17] This stems from two of the processes discussed above: (1) muscular weakness (disuse atrophy) and (2) contracture. As with the limb muscles, the muscles of respiration undergo atrophy with lack of use. This is especially true of the accessory muscles of respiration, including the intercostals. The ensuing weakness results in a loss of chest wall expansion. Lack of mobility also predisposes to loss of range of motion of the costovertebral and sternocostal joints through the process of contracture. Both the weakness of respiration muscles and the loss of mobility of the thorax lead to a restrictive reduction in pulmonary function. In addition, supine patients tend to hypoventilate.[194] Restriction of respiration gives rise to an elevated risk of atelectasis with a concomitant increase in arterial-venous shunting; this results in a perfusion of poorly ventilated regions,[195] leading to impaired oxygenation and fall in partial pressure of oxygen (PO_2) and rises in partial pressure of carbon dioxide (PCO_2) and pH. Immobilization has also been implicated in reduced ciliary activity in the upper respiratory tract, which impairs clearance of respiratory pathogens and increases the risk of pneumonia.[195]

Pulmonary Function Tests

The pulmonary function tests (PFTs) most directly affected by immobility are the vital capacity, tidal volume, minute volume, and maximal voluntary ventilation. This is not surprising, given the restrictive impairment caused by immobilization. Vital capacity and maximal voluntary ventilation can be reduced by more than one third from normal values.[17] Should such restrictive impairment be encountered, Hintzelmann has demonstrated that application of ultrasound diathermy to costovertebral joints followed by deep breathing exercises, result in improved PFTs in patients with ankylosing spondylitis.[196] This same technique can be applied to the patient who suffers unfavorable pulmonary effects due to immobilization. Therefore, PFTs should be performed at least once per week on patients at bed rest. Treatments included in pulmonary management of the immobilized patient are: (*a*) turn every 2 hours; (*b*) incentive spirometry every 4 hours while awake; (*c*) weekly PFTs; (*d*) assisted cough every 4 hours; (*e*) airway suctioning, if needed, every 4 hours; (*f*) percussion and postural drainage every 4 hours; (*g*) humidified air and mucolytic (acetylcysteine) inhalation for obstipated secretions four times daily; and (*h*) bronchodilators, if clinically indicated.

Lower Respiratory Tract Infection

The adverse effects of prolonged immobility are due primarily to gravitational effects on blood flow and ventilation, impairment of the normal mucociliary escalator, and possibly, an increase in extravascular lung water. The sequence of events that culminate in nosocomial pneumonia is unclear; however, low tidal volumes, increased extravascular lung water, and the accumulation of bronchopulmonary secretions may lead to atelectasis, a well-known precursor of pneumonia. Theoretically, continuous lateral rotational therapy should reverse these abnormalities.

In a randomized, prospective trial of 106 ICU patients, Fink and associates[197] tested the hypothesis that the incidence of lower respiratory tract infections in critically ill, blunt trauma victims can be significantly reduced by employing continuous postural oscillation. Among 48 patients in the control group, 47 met criteria for lower respiratory tract infection or pneumonia. By comparison, out of 51 patients in the treatment group, only 20 developed lower respiratory tract infection or pneumonia. It was concluded that continuous postural oscillation through a 40° to side arc for 10 to 16 hours per day decreases the risk of pulmonary sepsis in victims of major blunt trauma.

The efficacy of using lateral rotational treatment to prevent or reduce pulmonary complications in severely head-injured patients is unclear. Clemmer and colleagues[198] undertook a prospective, randomized trial which compared the kinetic treatment table to conventional bed care in severely head-injured patients. They found that there was no significant difference in mortality, CNS morbidity, length of stay, or rate of pulmonary complications between the two groups; this indicated that the efficacy of the kinetic treatment table in reducing pulmonary complications in head-injured patients remains unclear.

However, three prospective, randomized studies,[199-201] that evaluated patients with acute head trauma, stroke orthopedic injuries requiring traction, and blunt chest trauma, all showed a decreased incidence of nosocomial pneumonia with continuous lateral rotational therapy compared to those treated in a conventional bed and turned every 2 hours by the nursing staff. A fourth study,[202] performed in an ICU with a heterogeneous group of patients, showed no difference in incidence of nosocomial pneumonia between those treated with continuous lateral rotational therapy and those in a conventional bed, but it did show a decreased length of ICU stay for pneumonia patients treated with continuous lateral rotational therapy. It, therefore, appears that for continuous lateral rotational therapy to be effective, it needs to be instituted early in the patient's illness.[203]

The length of time that continuous lateral rotational therapy should be utilized is unknown. It is also unclear whether continuous lateral rotational therapy should be started at full rotation immediately or begun at lesser degrees of rotation and advanced serially over several hours. In the studies cited above, rotations from 40° to 62° in each direction were used. Another unknown is the minimum time that continuous lateral rotational therapy should be administered per day. In the studies discussed, most patients were rotated for 10 to 16 hours per day. Based on the current data, the early use of continuous lateral rotational therapy in comatose or otherwise immobile patients decreases the incidence of pneumonia over the first 7 to 14 days of ICU care.

Although the use of continuous lateral rotational therapy appears to be advantageous in the prevention of lower respiratory tract infections, its availability may be limited. If it is not available, the following is recommended: In addition to the every-2-hour turning program discussed above, aggressive pulmonary toilet should be performed at least every 4 hours. This includes assisted cough (diaphragmatic thrust in patients without abdominal or thoracic wounds), airway suctioning when necessary, chest physical therapy (percussion), and postural drainage. Incentive spirometry at least four times daily can help expand atelectasis and prevent costovertebral and costochondral contracure (Figure 12-8).[204] Humidified air and mucolytic medication such as acetylcysteine (Mucomist [Apothecon; Princeton, NJ]) will also be of benefit with opstipated secretions. If bronchospasm is present, use of broncho-dilators may be of additional benefit.

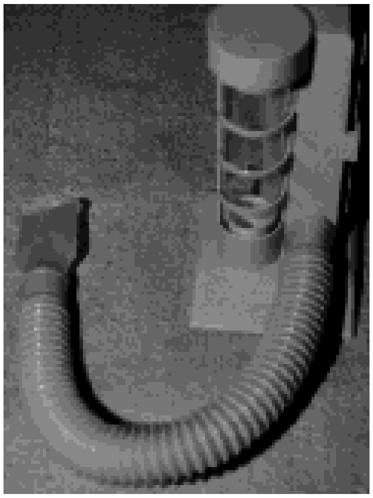


Fig. 12-8. Incentive spirometer for prevention of atelectasis. Maximum inspiratory effort should be performed every 4 hours while awake.

TABLE 12-4

CRITERIA FOR THE CLINICAL DIAGNOSIS OF PULMONARY EMBOLISM

	Likelihood of Pulmonary Embolism	
Clinical Findings	**High**	**Low**
Reasonable precipitating cause	Yes	No
Typical symptoms and signs	Several	Few
$Pa_{O_2} < 80$ mm Hg	Yes	No
FDP and SFC	Both positive	Both negative
Chest roentgenogram	Abnormal	Normal

FDP: fibrin degradation products
SFC: soluble fibrin complexes
Pa_{O_2}: partial pressure of oxygen, arterial
Source: Wilson JE. Pulmonary embolism. In: Wyngaarden JB, Smith LH, Bennett JC, eds. *Cecil Textbook of Medicine*. 17th ed. Philadelphia, Pa: WB Saunders; 1985:426-431.

Pulmonary Embolism

Pulmonary embolism is a serious medical complication with life threatening consequences. Up to 20% of all deaths occurring in hospitals are a result of pulmonary embolism.[205] The immobilized patient is at particular risk. Xue and Zhang used rabbits to develop an experimental model for inducing pulmonary fat embolism by forced immobilization.[206] Forced immobilization for 5 hours was found to induce pulmonary embolism. The results suggest that the disorder of homeostasis caused by immobilization alone may bring about pulmonary embolism. In one autopsy study,[207,208] pulmonary embolism was shown to be present in 50% of patients who die in hospital; but less than 50% of the patients who died were suspected of having pulmonary embolism prior to death.

The origin of pulmonary embolism results from thrombi that migrate and pass through the right atrium and ventricle and lodge in the pulmonary arteries. (The reader is referred to the section on venous thrombosis for its etiology.) Approximately 50% of patients with diagnosed pulmonary embolism have no clinical sign of deep venous thrombosis.[209] Yet, between 85% to 95% of all pulmonary emboli are believed to arise from lower extremity thrombi.[210] The resulting ventilation-perfusion mismatch results in reduced PO_2 with resulting complaints of dyspnea. Substernal crushing pleuritic chest pain and hemoptysis may or may not be present. Tachycardia and tachypnea are often observed. Cyanosis may also be noted if the hypoxia is significant. Differential diagnosis includes pulmonary infection (pneumonia), atelectasis, congestive heart failure, acute myocardial infarction, dissecting aortic aneurysm, ruptured esophagus, bronchospasm, and mucous plugging. Table 12-4 lists criteria for the clinical diagnosis of pulmonary embolism.[205]

Chest roentgenograms remain useful in diagnosis, particularly if infarction has resulted in a wedge-shaped infiltrate, but the diagnosis can be suspected from roentgenograms in only about 50% of patients with pulmonary embolism (Figure 12-9).[211] Use of radioisotope ventilation-perfusion scanning can further assist with diagnosing suspected pulmonary embolism, although the diagnosis is definitively made through pulmonary angiography (Figures 12-10, 12-11, and 12-12).[212] It must be noted that there is a 4% to 10% risk of morbidity associated with pulmonary angiography. The acceptable diagnostic endpoints for diagnosis of pulmonary embolism without angiography are as follows[205]:

1. Pulmonary embolism is excluded
    - normal perfusion scan
    - low probability perfusion scan with:
        - low estimate of clinical likelihood of pulmonary embolisms, or
        - normal chest x-ray
2. Pulmonary embolism is confirmed
    - high probability perfusion scan with:
        - high clinical likelihood, or

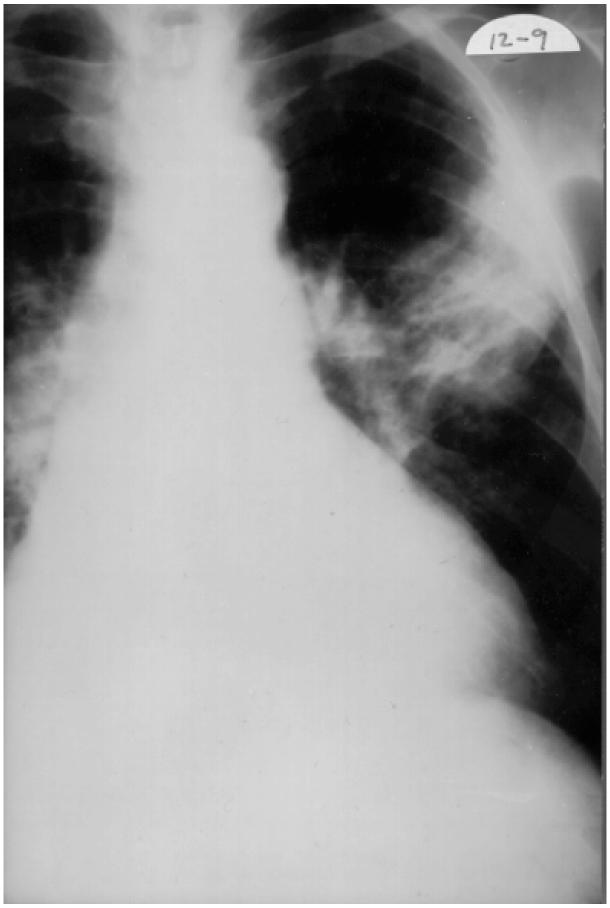


Fig. 12-9. Chest radiograph showing a left wedge-shaped pulmonary infiltrate in pulmonary embolism. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

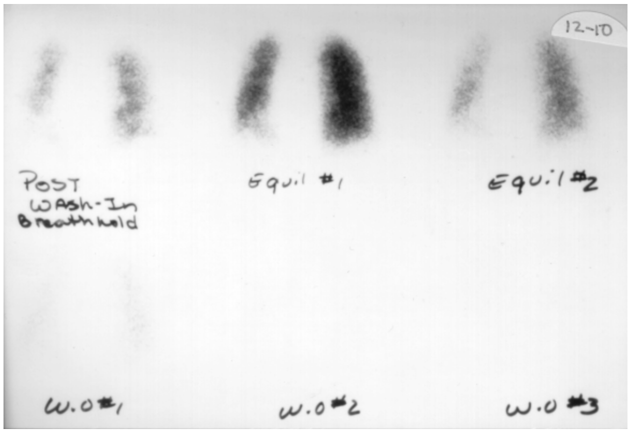


Fig. 12-10. Complete ventilation of all lung fields. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

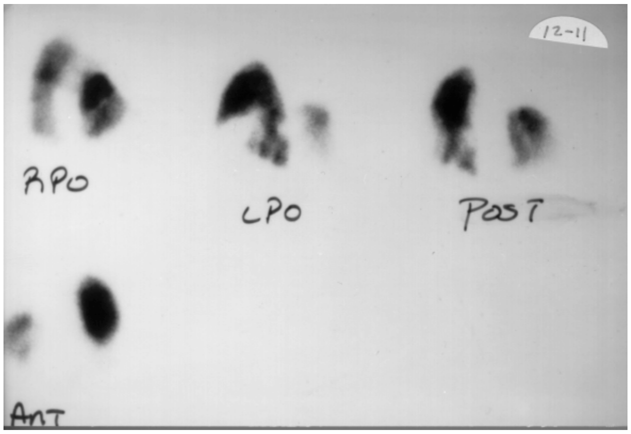


Fig. 12-11. Impaired perfusion of the left lobe of lung. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

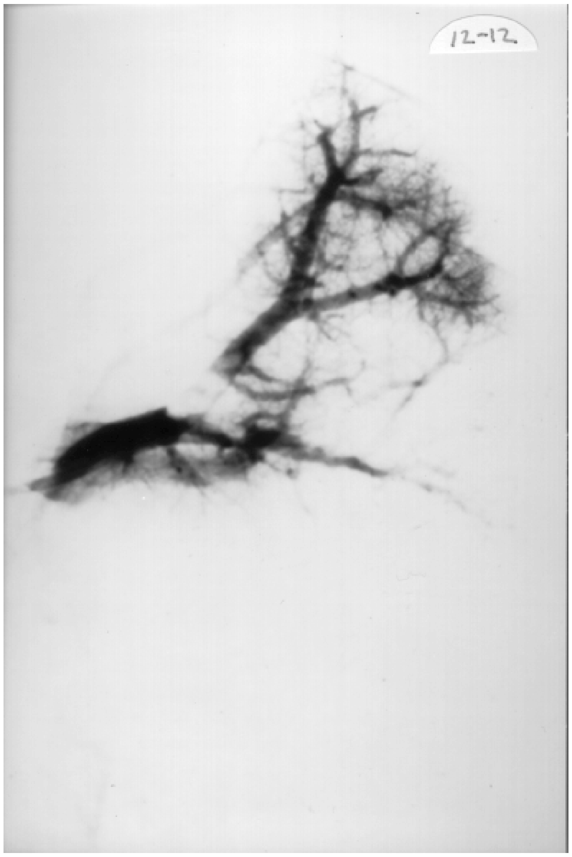


Fig. 12-12. Pulmonary angiogram demonstrating massive left pulmonary embolism. The lower lobe artery is totally occluded, and there is a large filling defect in the artery to the upper two segments. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

- normal ventilation scan, or
- positive venogram, or
- impedance plethysmography.

Thirty-three percent of deaths from pulmonary embolism occur within the first hour of the embolism lodging in the pulmonary arteries, thus making prevention the most important management strategy. Prevention of pulmonary embolism has been investigated through institution of early heparin therapy, including patients with spontaneous intracerebral hemorrhage.[213] In 68 patients with spontaneous intracerebral hemorrhage, the effect of low-dose heparin treatment beginning on the 2nd, 4th, or 10th day was investigated. Early (day 2) low-dose heparin medication significantly lowered the incidence of pulmonary embolism. An increase in the number of patients with rebleeding was not observed. The results indicate that the early use of heparin in these patients is safe and can be recommended for the prevention of thromboembolic complications. The recommended dose is 5,000 units, subcutaneously, twice daily.[205] For additional preventive measures, the reader is referred to the section on venous thrombosis.

For confirmed pulmonary embolism, anticoagulation is necessary.[214] The duration of treatment should be at least 3 months.[205] Fibrinolytic therapy has also been used to dissolve emboli. The use of thrombolytic therapy is reserved for life-threatening situations.[205,215] If anticoagulation therapy is inadequate or contraindicated, partial venous interruption may be indicated and accomplished through placement of an intraluminal filter in the inferior vena cava.[216] Heroic measures include pulmonary embolectomy.[217]

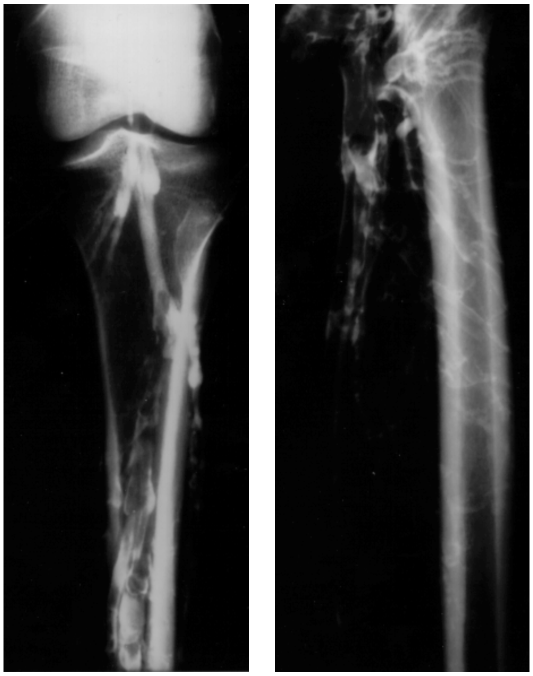
Early mobilization reduces the risks of all pulmonary complications, including pulmonary embolism. When immobilization is necessary, following the guidelines set forth above should help minimize these risks.

Cardiovascular System

Thromboembolism

It is estimated that there are nearly 20 million cases of lower extremity deep venous thrombosis in the United States annually.[218] Thromboembolism is known to occur in the immobilized patient.[219] One study[220] has attributed up to 25% of acute iliofemoral venous thrombi directly to immobilization. If additional risk factors are present, the danger is magnified. The risk factors for lower extremity deep venous thrombosis include prolonged inactivity, trauma (including long bone fractures), paralysis or paresis, dehydration, advanced age, obesity, malignancy, hypercoagulable states, pregnancy, premenopausal estrogen use, polycythemia, and congestive heart failure.[209]

In a study of immobilized multiple trauma patients, 60% had clinically silent, deep venous thrombosis, with half of these extending above the knee.[221] Venous thrombosis develops when the components of Virchow's triad are present: stasis of blood flow, hypercoagulable state, and endothelial damage. In the immobilized patient all three of these are possible. Most venous thrombi seem to originate in regions of slowed blood flow, particularly in the veins of the calf and thigh (Figures 12-13 and 12-14). Decreased blood flow, or even stasis due to lack of the pumping action of immobilized muscles, is undoubtedly a major factor. As blood pools, activation products of the coagulation system accumulate and lead to local hypercoagulability. Activation products of clotting and fibrinolysis can induce



Figs. 12-13 and 12-14. (**12-13,** left) Venogram demonstrating a popliteal deep venous thrombosis. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC. (**12-14,** right) Venogram demonstrating a femoral deep venous thrombosis. Reprinted from the Teaching File, Department of Radiology, Walter Reed Army Medical Center, Washington, DC.

endothelial damage, which, in turn, leads to further activation of the hemostasis system. Endothelial damage may also result from distension of the vessel walls by the pooling blood. Blood flow is further decreased by hyperviscosity due to elevated fibrinogen levels and dehydration.[222]

Deep venous thrombosis in the popliteal vein, or proximally, are felt to be more dangerous than distal thrombosis due to the higher risk of pulmonary embolism.[210] More than half of all patients with a documented deep venous thrombosis will have at least a clinically silent pulmonary embolism.[223] The percentage for calf deep venous thrombosis is less, but still in excess of 15%.

Clinical suspicion should be high for any patient at bed rest for more than a few days. Signs of possible thrombosis include unilateral edema of an extremity, pain, erythema, palpable cords in the calf, or Homan's sign. Homan's sign is calf pain with passive dorsiflexion of the foot with the knee slightly flexed (to relax the gastocnemii). Of these indicators, only unilateral swelling of the leg, either above or below the knee, is a reliable discriminatory sign of venous thrombosis.[210] Therefore, regular measures of calf and thigh circumference are essential in monitoring the immobilized patient for venous thrombosis. Unfortunately, Homan's sign is present in only 10% of documented cases of deep venous thrombosis.[209] It is also nonspecific when present, as it can be present in any cause of calf inflammation or herniated low lumbosacral intervertebral disk.[224]

Although the differential diagnosis of deep venous thrombosis is extensive, it can be organized into general categories.[225] These include soft tissue inflammatory disorders (cellulitis, myositis, tendonitis), popliteal (Baker's) cyst, trauma, neoplasm, other venous disorders (varicose veins, superficial thrombophlebitis, extrinsic venous compression), bone disorders (fracture, heterotopic ossification, osteomyelitis), peripheral arterial disease, arthritis, peripheral nerve disorders (including radiculopathy), systemic edema (congestive heart failure, cirrhosis, malnutrition, pregnancy), and lymphedema. A detailed listing of the differential diagnosis is available in a text devoted to venous thrombotic disorders.[226]

Contrast venography has been the standard on which all noninvasive techniques for detecting deep venous thrombosis are compared (see Figures 12-13 and 12-14). Although this technology is extremely sensitive and specific, its disadvantages include discomfort, contrast reactions, and relative expense.[210] Visualization of the iliac veins can be problematic because of rapid dilution of contrast in the these large veins.[227] Of note, venography itself can cause deep venous thrombosis in up to 3% of patients undergoing the test.[228] Impedence plethysmography, a noninvasive test which measures blood volume in the leg by changes in electrical impedence, is sensitive and specific for detecting proximal lower extremity deep venous thrombosis. It is quick to perform and relatively less expensive than contrast venography. It is, however, inaccurate for detecting distal (calf) deep venous thrombosis.[210]

Color-flow Doppler ultrasonography is a recent advance in using ultrasound in the evaluation of deep venous thrombosis. Blood flow away from the transducer appears blue, while flow toward it is red. The intensity of the color is proportional to the flow rate. Stationary structures, such as vessel walls, appear gray. Compared to venography, the sensitivity and specificity of this test approaches 100% for proximal deep venous thrombosis and shows promise for the noninvasive diagnosis of distal (calf only) deep venous thrombosis.[210,229,230] A diagnostic algorithm is presented in Figure 12-15.[227]

Thrombosis prophylaxis should reduce the incidence of this potentially life-threatening complication. Simple steps that can be taken include elevation of the legs and use of thigh-high graduated-compression elastic stockings while the patient is at bed rest.[225,231] Intermittent pneumatic calf compression may be a useful treatment to substitute for the reduced venous pumping in the lower extremities.[231,232] Isometric exercise of the lower extremities will serve the same purpose. A further recommended prophylactic measure is 5,000 units of heparin subcutaneously twice daily until the patient is remobilized. If a deep venous thrombosis is diagnosed, then anticoagulation is indicated. Traditionally, this has meant intravenous heparinization to achieve an activated partial thromboplastin time of between 1.5 and 2.5 times the control value, and initiation of warfarin sodium by the second treatment day.[233] When the prothrombin time (PT) reaches two times control, the heparin is discontinued (typically 5 days), and warfarin is maintained for 3 months with a PT between 1.5 and 2.0 times control.[234-238] PT should be checked three times a week during the first week of therapy, twice weekly for the next 2 weeks or until a stable dose of warfarin is achieved, then weekly thereafter.[236,237]

A clinical trial[239] has found that replacing continuous intravenous heparin with subcutaneously administered low molecular weight heparin, dosed at 175 international factor Xa inhibitory units per kilogram of body weight once daily is an equally

Fig. 12-15. Diagnostic algorithm for the diagnosis of deep venous thrombosis (DVT). Reprinted with permission from Satiani B, Rustin R, Biggers K, Bordner L. Noninvasive diagnosis of deep venous thrombosis. *Am Fam Physician*. 1991;44(2):569–574. © American Academy of Family Physicians. All rights reserved.

effective treatment for proximal vein thrombosis and allows outpatient treatment of uncomplicated thrombi. Thrombolytic treatments have also shown promise, especially in preventing symptoms of postphlebitic venous insufficiency.[215] Inferior vena cava interruption is reserved for patients who develop pulmonary emboli despite adequate anticoagulation or for those who cannot be anticoagulated.[240]

Cardiovascular Deconditioning

Among the major cardiovascular changes that occur with immobilization are fluid shift, negative fluid balance, orthostatic instability, decreased exercise tolerance, and loss of blood volume.[241,242] With bed rest, there is a redistribution of body fluids from the lower extremities to the central circulation. This fluid shift can be in excess of 0.5 L,[6,243] and affect homeostasis. This has two major consequences:

1. The heart rate decreases in response to increased central venous pressure, increased left ventricular end diastolic volume, and increased stroke volume. Initially, this results in up to a 40% increase in cardiac output in the supine position.[244] Cardiac output is reduced toward baseline levels through reduced heart rate, and blood pressure is controlled through reduced peripheral resistance.[241-243] With prolonged bed rest, there is a gradual increase in the resting heart rate of an average of 1 beat per minute every 2 days up to an overall increase of up to 15 beats per minute above basal.[241,242]

2. The central venous pressure is reduced through negative fluid balance, or excretion. During the first week of bed rest there is a corresponding plasma volume loss of approximately 7%.[245] There is a parallel loss of blood volume, with a resultant 8% decrease in red blood cell mass.[245] Although these losses stabilize, the result is relative dehydration.

The impact of these hemodynamic changes becomes apparent when, after weeks of bed rest, the immobilized subject stands. On standing, the heart rate increases by nearly 30% of that of a mobile subject. Despite the increased pulse, cardiac output falls by a similar degree.[245] And although peripheral vascular resistance does increase, it is not sufficient to compensate for the reduced stroke volume and the blood pressure falls on standing. This orthostatic intolerance is manifested clinically by tachycardia, diaphoresis, nausea, and possible syncope.[242] Postural hypotension occurs when the pulse increases by 20 beats per minute and the systolic blood pressure falls by more than 20 mm Hg on rising from a supine position.

Another major consequence of prolonged bed rest is reduced aerobic capacity, or exercise tolerance, as a result of decreased volume of maximum oxygen consumption (VO_2max). VO_2max is a function of the body to deliver oxygen to the tissues and is a product of maximal cardiac output and maximum arteriovenous oxygen difference. Because cardiac output does not increase in response to exercise to the same degree as it does in the nonrested subject, reductions of up to 46% in VO_2max have been reported following 4 weeks of bed rest.[241] Decrease in VO_2max occurs whether the patient is at complete bed rest or is allowed to exercise in bed.[246] However, this loss of VO_2max can be minimized by performing isometric exercise.[247] Finally, it has been recently observed that left ventricular atrophy occurs in bedridden elderly patients.[248]

In attempting to mobilize a patient who has been at bed rest for more than 3 to 5 days, it is important to monitor blood pressure and pulse to observe if hypotension is occurring. The signs of orthostasis mentioned above should be looked for. It may be necessary to use a passive tilt table, starting with only a small angle of incline for relatively short periods of time, to reacclimate the patient to the nonhorizontal position. The rapidity of progress is dependent on the patient's tolerance. The longer he or she has been at bed rest, the longer this process will take.

Vallbona[17] has suggested a regimen that starts with the table at 30° of incline from the horizontal, for 1 minute, twice a day and builds tolerance at that angle gradually until this angle can be tolerated for 30 minutes twice a day. Once this is accomplished, the angle can be advanced 5° to 10° per week until an angle of 70° is reached, which approximates 1G of force. At 1G of force the body experiences the normal acceleration force of gravity in the upright position (approximately 9.8 m/s^2).[249] From this point, progressive ambulation can begin.

While on the tilt table the patient will need to be monitored closely for signs of orthostatic hypotension. Several measures can be added to assist in mobilizing the patient and prevent orthostasis. To prevent venous pooling of blood in the lower extremities, thigh-high graduated-compression elastic stockings should be applied. An abdominal binder can also assist in maintaining venous return to the heart. Should either of these measures prove ineffective, adrenergic support in the form of ephedrine, 25 mg by mouth, 20 to 30 minutes before therapy, can be of assistance.

Once the patient is able to tolerate the upright position, a program of cardiac reconditioning can begin. Initially, exercise activity should be limited to 70% of the maximal heart rate (roughly 70% of 220 minus age) or an elevation in resting pulse of 20 beats per minute to begin aerobic conditioning.[56] The duration of the exercise and how rapidly it can be advanced will depend on patient tolerance. Maximum gains in endurance and aerobic capacity will be on the order of 20% to 40% per week. The program should include both warm-up and cool-down exercises to prevent exercise induced ST segment depression and postexercise hypotension.[56]

CONCLUSION

The era in medical history of employing complete bed rest as a primary treatment has passed. The complications of immobility generate staggering costs in healthcare dollars and resources, and lost productivity can be substantial.

Implications for extended loss of personnel in a combat situation, given the multisystem physiologic effects and complications of immobilization, are not inconsequential, but prevention of these complications can have a substantial impact on the practice of military medicine.

All the complications described in this chapter can be minimized, if not prevented, by avoiding the use of immobilization and bed rest as much as is practical. When necessary, immobilization should be for as small a region as possible, for the shortest amount of time possible. There are very limited uses for prolonged immobility. When a patient is prescribed bed rest, it is the responsibility of the care providers to be vigilant and aggressive with interventions to prevent the complications that will inevitably result from the altered homeostasis. Physical medicine services can be of great assistance in evaluating, educating, managing, reconditioning, and preventing the ill effects that can befall the immobilized patient.

ACKNOWLEDGMENTS

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Chapter 13

PHYSICAL FITNESS AND PHYSICAL TRAINING FOR MILITARY PERFORMANCE

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INTRODUCTION

STRUCTURE AND FUNCTION OF SKELETAL MUSCLE
  Muscle Structure and Contractility
  Neural Control of Muscle Activity
  Energy Transformation Within the Muscle

PRINCIPLES OF EXERCISE PHYSIOLOGY
  Energy Transfer in Exercise
  Pulmonary Function During Exercise
  Cardiovascular Function During Exercise

BODY COMPOSITION AND PHYSICAL FITNESS
  Background
  Physical Training
  Body Composition and Physical Capacity
  Body Composition Standards
  Body Fat Methodology

PHYSICAL CAPACITY FOR EXERCISE
  Aerobic Power
  Anaerobic Power
  Muscular Strength

PHYSICAL CAPACITY OF MILITARY POPULATIONS
  Aerobic Capacity
  Muscular Strength and Muscular Endurance

PRINCIPLES OF PHYSICAL TRAINING
  Training Principles
  The Training Response
  Training Program Components

PHYSICAL TRAINING PROGRAMS
  General Considerations
  Aerobic Training
  Anaerobic Power Training
  Strength Training
  Training Program Integration
  Occupational or Mission Specific Physical Training

MEDICAL PROBLEMS ASSOCIATED WITH PHYSICAL TRAINING AND EXERCISE
  Musculoskeletal Injuries
  Cardiovascular Disease
  Exertional Rhabdomyolysis
  Environmentally Caused Injuries

CONCLUSION

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INTRODUCTION

Despite the rapid modernization of weapon systems and technological advancements in war-fighting capability, the military profession remains physical in nature. Considerable emphasis is placed on physical training, physical capacity, and ultimately, on physical readiness, regardless of the specific role or occupation that the service member may fill. Irrespective of peacetime or wartime, service members must be prepared to defend themselves and others about them, and to react to emergencies as they may occur.

Like other organizations dealing with emergencies, such as police and firefighters, the military services emphasize physical fitness for purposes of discipline, morale, health maintenance, and physical appearance in addition to the traditional role of meeting the physical demands of the job. Thus, despite the many changes toward automation, mobility, and weapons of mass destruction, the military remains a physical force with an emphasis on high levels of fitness through formal physical training programs. Fitness was highlighted and emphasized to all the military services in 1981 when the Department of Defense (DoD) issued a new directive on physical fitness and weight control. The directive states:

> Physical fitness is a vital component of combat readiness and is essential to the general health and well-being of armed forces personnel. Individual members must possess the stamina and strength to perform successfully any potential mission. These qualities, together with weight control, form the basis of the DoD physical fitness program.[1]

An understanding of physical fitness, and physical performance and its development through physical training should be an integral part of the training of military physiatrists. An understanding of normal muscle and exercise function must be present before one can repair, treat, and rehabilitate the injured.

The term "physical fitness" is an often abused term that requires some attention and definition. Ability to perform physically demanding tasks is a function of two groups of factors: (1) factors that determine the capacity for muscular contraction, and (2) factors that relate to the neural control of body movement. The latter, which may be most correctly referred to as *motor fitness*, includes the components of neuromuscular control, such as coordination, speed, agility, and skill, which are achieved primarily through repeated practice. The first group of factors, commonly referred to as *physical fitness*, are those associated with the energy generating capacity for muscular exercise. *Physical fitness* is developed through physical-training-induced physiological adaptations and will be the primary emphasis of this chapter. Thus, we define and use the term physical fitness as the *energy generating capacity to perform physical effort*.

Three distinct, but overlapping, energy generating systems for muscular exercise form the common categories of physical fitness: (1) stored energy located in the muscle cell mitochondria in the form of high energy phosphagens that are associated with *muscle strength*, (2) rapidly produced energy in the form of phosphagens generated by the anaerobic process of glycolysis and associated with *muscular endurance* (or muscular power or anaerobic power), and (3) more slowly produced energy in the form of phosphagens derived from the aerobic metabolism of various substrates and associated with *aerobic power* (or cardiorespiratory endurance). Because each category of fitness involves a distinctly different energy generating metabolic pathway, each also requires different approaches for development through training. This chapter will discuss the capacity for exercise, physical performance, and training adaptations in the context of these three categories of physical fitness or energy generating systems.

STRUCTURE AND FUNCTION OF SKELETAL MUSCLE

Physical activity is the result of skeletal muscle producing force through the process of contraction. This contraction, which produces a pulling action against bone, causes a body segment to move about its joint axis and thereby produces movement. Body movements are actually the result of the coordination between the muscles that are shortening (contracting) and those that are opposing the movement by relaxing (referred to as the antagonist muscles). Most muscular activity consists of the highly complex coordination of a number of muscles or muscle groups.

When muscles contract, they do not always shorten. There are three types of muscular contrac-

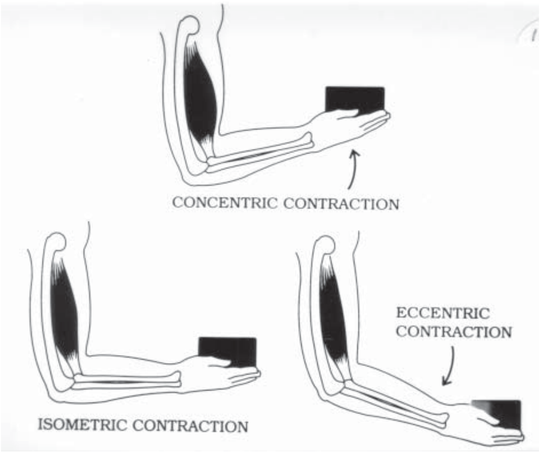

Fig. 13-1. The three types of muscular contractions.

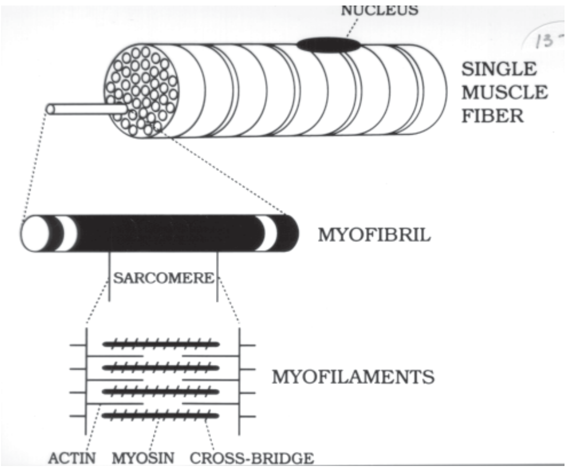

Fig. 13-2. Structure of the skeletal muscle fiber.

tion: (1) shortening (*concentric*) contractions, (2) static (*isometric*) contractions, and (3) lengthening (*eccentric*) contractions. Many movements, such as walking or lifting and lowering a weight, will involve all three types. These are illustrated in Figure 13-1. Only in the first case, concentric contraction, does the muscle actually shorten. In the case of a static contraction, the force generated equals the resistance to movement and, therefore, no movement takes place. In eccentric contractions, the external resistance overcomes the force developed by the muscle and the muscle is actively stretched, such as in the controlled lowering of a weight to the floor.

Muscle Structure and Contractility

Skeletal muscle is composed of muscle tissue, as well as its nerve and vascular supply, and some connective tissue, and is attached to bone at each end by tendons. The muscle tissue itself is composed of muscle cells, commonly referred to as muscle fibers due to their long and slender shape. The structural makeup of the muscle fiber is illustrated in Figure 13-2.

Each muscle fiber is composed of numerous myofibrils lying parallel to each other along the long axis of the fiber. Each myofibril is composed of a series of sequential identical units referred to as sarcomeres. Each sarcomere can independently contract. Simultaneous contraction of all sarcomeres results in tension being generated by the fiber. Each sarcomere, in turn, is composed of two types of protein filaments: a thicker filament of the protein myosin and a thinner filament of the protein actin. The partial overlapping of these filaments causes the appearance of bands on the myofibril.

Muscular contraction is produced by the sliding of the thin filaments past the thicker filaments in a telescopic manner as illustrated in Figure 13-3, thus shortening the sarcomere. Although not precisely known, it is believed that the sliding of the filaments is caused by protruding structures from the myosin molecules called cross-bridges. These cross-bridges have an affinity for actin and contain the enzyme, myosin adenosine triphosphatase (ATPase), which hydrolyzes high energy phosphates to release their energy for the contractile process. In the resting state this affinity, and therefore the interaction between the two filaments, is blocked by two additional proteins (troponin and tropomyosin) found in the thin filaments. This inhibition is released by calcium ions (Ca^{++}). As Ca^{++} binds to troponin and tropomyosin, the actin is freed to bind

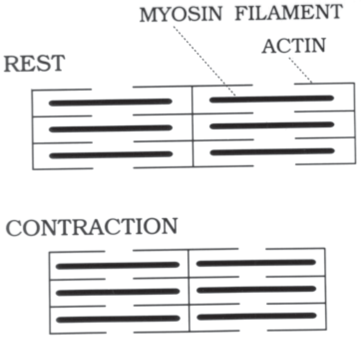

Fig. 13-3. Depiction of the sarcomere shortening process during muscular contraction.

with myosin. The energy then released through the hydrolysis of adenosine triphosphate (ATP) causes the cross-bridges to pull the filaments past each other, possibly in a ratchet-like motion. Muscle contraction is initiated by an increased concentration of Ca^{++} in the myofibril to initiate this chain of events. The reader is referred to Astrand and Rodahl[2] for further discussions of the mechanisms of sarcomere shortening and force development within the muscle fiber.

Fiber Types

Skeletal muscle fibers are not homogenous. Human skeletal muscle is composed of two distinct populations of fibers that differ in terms of chemical makeup and contractile characteristics: type 1, commonly referred to as slow twitch fibers; and type 2, fast twitch fibers. Type 2 fibers may also be divided into two or three subtypes.

Type 1 fibers are functionally characterized by a relatively long buildup time to peak tension (80-100 ms). This build up is the result of the low number of fibers innervated by each neuron (motoneuron). Type 1 fibers have high mitochondrial and capillary densities that result in their high capacity for oxidative phosphorylation. Thus, they are particularly suited for aerobic activities and are relatively resistant to fatigue. Type 2 fibers, in contrast, are characterized by fast rise time to peak tension (40 ms), and having a high fiber-to-motoneuron ratio, reach relatively high peak tensions. These fibers are characterized by high concentrations of the anaerobic glycolytic enzymes, such as ATPase. They are particularly suited for brief, intense periods of activity but are rapidly fatigued.

Type 2 fibers can be further divided into the subtypes 2a and 2b, based on their enzyme content and activity. Type 2a fibers tend to be biochemically intermediate between 2b and 1. The reader is referred to the review by Saltin and Gollnick[3] for more detailed descriptions of fiber characteristics. These fiber types can be distinguished by the strength with which the enzyme myofibrillar ATPase binds to myosin as detected with histochemical staining (Figure 13-4).

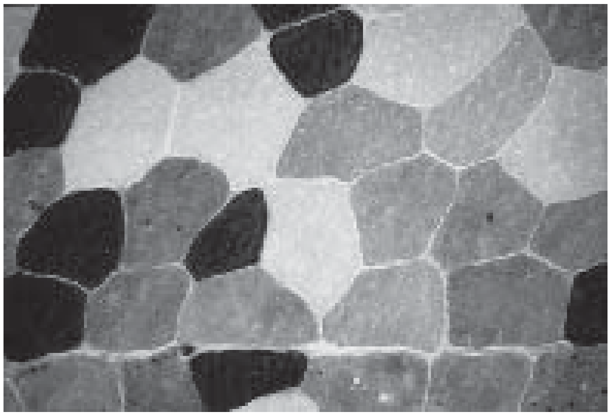

Fig. 13-4. Muscle cross-section showing different fiber types by the density of histochemical staining for ATPase. Darkest fibers are type 2, lightest are type 1, and intermediate density are types 2a and 2b.

Fiber Type Distribution

The relative distribution of type 1 and 2 fibers varies between muscle groups and between individuals. While major muscles of propulsion, such as the vastus lateralis and gastrocnemius, are, on average, approximately evenly divided between the two fiber types, postural muscles, such as the soleus that sustain tension over long periods, have about 70% of type 1. While muscles such as the vastus lateralis are, on average, evenly distributed between the two types, individual differences range from 10% to 95%.[3] For example, high ratios of type 1 are often found in elite endurance athletes, while elite sprinters and weight lifters typically show high type 2 fiber counts. Their athletic success appears to be related in part to their natural endowment with a particular fiber type. Fiber typing has been employed to predict potential success in a particular sport.

The proportion of fiber types in an individual is genetically determined. Fiber type is dictated by its innervation, which is established early in fetal development. A particular motoneuron innervates only one type of fiber. A fiber retains its nominal characteristics throughout life, and, therefore, the relative distribution of types remains unchanged. Intense physical training will cause a fiber's chemical and contractile characteristics to partially shift toward that favored by the type of training, but this shift is temporary and lasts only as long as the training stimulus is continued.[3]

Neural Control of Muscle Activity

Muscle Innervation

Voluntary skeletal muscle function is controlled by the motor unit. The motor unit is composed of the anterior (alpha) efferent motoneuron cell; its

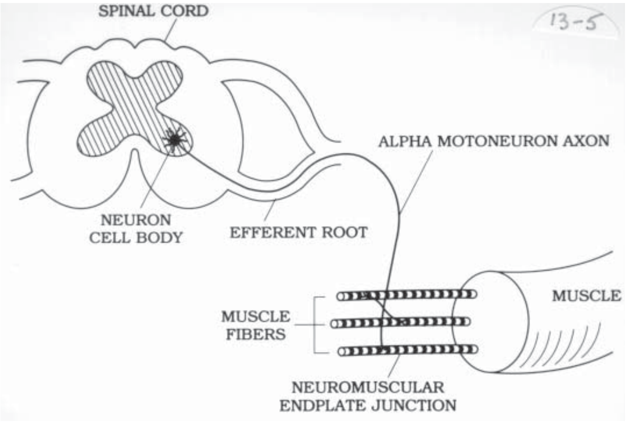

Fig. 13-5. The motor unit.

axon, the motor endplate, and the muscle fibers innervated by it (Figure 13-5). Each neuron innervates multiple fibers, but all of the same type. Muscles involved in fine movements have low neuron-to-fiber ratios, while muscles concerned with gross movements have high ratios. The efferent motoneuron has a corresponding sensory afferent nerve unit consisting of a muscle spindle (sensory receptor) and sensory neuron that synapses with interneurons in the spinal cord, which in turn, synapse with the efferent neuron. This is referred to as the *reflex arc*. The reflex arc provides the feedback and control that many physical movements require.

To produce muscle contraction, the nerve impulse emanates from the brain or spinal cord and travels along the neuron to the muscle across an interface referred to as the motor endplate or neuromuscular junction. Transfer of the electrical signal from the nerve across the junction is accomplished by the chemical (neurotransmitter), acetylcholine. Acetylcholine is stored in, and then released by the nerve signal from, sac-like vesicles at the end of the terminal axons. Acetylcholine released into the interface enters the postsynaptic membrane on the surface of the muscle fiber, causing it to depolarize by allowing the movement of sodium and potassium ions. Depolarization then spreads along the entire fiber to produce the contraction. Repolarization begins when the acetylcholine is destroyed by the enzyme cholinesterase. The end products are used to resynthesize acetylcholine.

Force Development

A single nerve impulse along the alpha motoneuron will produce an action potential that spreads to all of the innervated fibers resulting in a twitch, a rise in muscle tension followed by relaxation. If consecutive action potentials reach the muscle before relaxation is complete, contraction will occur again, but it will start at a higher level and rise each time to a higher tension, called *summation*. At high rates of stimulation, no relaxation occurs and the tension remains constant (tetanus), creating a state of mechanical fusion of the contractions (Figure 13-6).

Initial tension produced during stimulation of a muscle must first overcome the resistance produced by certain components of the muscle, particularly the connective tissue, tendons, and elasticity in the cross-bridges. This resistance is referred to as the *series elastic component* and must be overcome before external force is produced. The series elastic component is comparable to a spring that must be stretched before an action takes place.

Muscle Control

Neural control of muscle contractile force can be brought about in two ways: (1) by adjusting the frequency or the pattern of stimuli and (2) by adding additional motor units. As pointed out earlier, increasing the rate of action potentials along a motor unit will produce increased tension up to tetanus. The more important mechanism, however, is through the addition of more motor units, referred to as *motor recruitment*. High muscle forces can be generated by bringing into play numerous motor units. The production and regulation of tension is a complex function of integrating the rate of action potentials, recruitment of motor units, the type of fibers being recruited, and the synchrony of the firing pattern. For example, in weight lifting there is typically a synchronous pattern (simultaneous firing) of fast twitch motor units firing. Running, on the other hand, is more typically asynchronous, where some units, predominantly slow twitch, are firing while others are recovering.

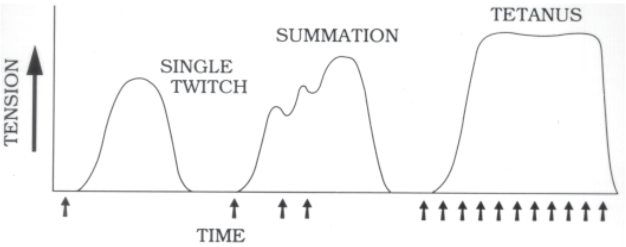

Fig. 13-6. Muscular tension during twitch, summation, and tetanus.

Muscle Fatigue

The term *fatigue* might best be defined, in reference to physical activity, as the loss of muscular power or exercise capacity. If maximal voluntary force is exerted, that maximal force output can only be maintained for a few seconds before the fiber mitochondria are depleted of their phosphagen stores and the tension begins to decrease, thus, "fatigue." If an individual runs at the level of his maximum oxygen consumption (VO_2max), that exercise intensity can only be maintained for 5 to 10 minutes after which he must stop or reduce the intensity level, again referred to as a loss in capacity or fatigue.

The cause and location of fatigue depend on the nature of the muscular activity. In most cases, the contractile mechanism within the muscle is implicated, rather than the neural signal to the muscle. In some cases of very intense effort when all motor units are presumably activated, decreases in action potentials or failure for the action potential to cross the neuromuscular junction have been detected. More common, however, is the situation where the neural impulse to the muscle is maintained, but failure occurs in the fibers. This loss in contractile tension in the fibers is most often the result of either insufficient energy substrate or insufficient blood flow to the muscle tissue. The latter impairs oxygen (O_2) delivery to the cells and the removal of the metabolic end-products, water, and heat. These conditions will interfere with the contractile mechanism of the myofibrils by preventing the repletion of the high energy phosphate stores, interfering with the energy transfer process, or disrupting the depolarization of the myofibrils, or both. In the case of an intense isometric contraction, pressure builds in the tissue through accumulation of fluid so that tissue pressure exceeds arterial pressure and impedes blood flow in and out of the muscle, resulting in anoxia and failure of the fibers to maintain tension. Causes of fatigue in rhythmic and whole body exercise will be discussed in later sections.

Energy Transformation Within the Muscle

As already pointed out, the energy required to cause the filaments to slide together for muscle to shorten and produce tension is derived from high energy phosphate bonds provided by ATP. ATP consists of a molecule of adenosine linked to three phosphate groups. The two linking bonds of the outermost group are referred to as high energy bonds because of their high potential energy. This potential energy is released to myofibril proteins when ATP is hydrolyzed, breaking the outer bond, and leaving adenosine diphosphate (ADP). This energy can be released immediately from ATP in the cells' mitochondria without the presence of O_2. Such stored ATP is the immediate source of energy for the initiation of exercise or a brief exercise bout, such as the lifting of a weight. However, the amount of stored ATP is sufficient for only a few seconds of maximal contractions. For muscle contraction to continue, the ATP must be resynthesized. Energy for this resynthesis comes from another energy-containing phosphate compound, creatine phosphate (CP), which is much more plentiful in the cell.

Hydrolysis of CP provides the energy for the regeneration of ATP from ADP. Energy for this transfer of phosphate bonds (phosphorylation) comes from the oxidation of carbohydrates, fats, and proteins in consumed foods. During this oxidation process, hydrogen atoms are removed from these nutrient substrates. Within the cells' mitochondria, electrons are removed from these hydrogen atoms and passed by electron receptors to molecular O_2 to produce the energy for ATP resynthesis. These electron receptors are the coenzymes, nicotinamide adenine-dinucleotide (NAD^+) and flavin adenine dinucleotide (FAD). These coenzymes are found in the B vitamins, niacin and riboflavin, respectively. They each gain hydrogen and electrons to become NADH and $FADH_2$, respectively. Electrons carried by these molecules are then passed along through a series of five iron-protein electron carriers, the cytochromes. The last cytochrome passes the electrons to molecular O_2. During this electron transfer process, chemical energy is trapped in the formation of the high energy phosphate, ATP. Over 90% of ATP formation takes place during this electron transfer process, referred to as oxidative phosphorylation. The reaction can be represented as follows:

$$\text{NADH} + \text{H}^+ + 3\ \text{ADP} + 3\ \text{P} + \tfrac{1}{2}\ \text{O}_2 \longrightarrow \text{NAD}^+ + \text{H}_2\text{O} + 3\ \text{ATP}$$

It is thus apparent that for the resynthesis of ATP and muscular contraction to proceed beyond a few seconds, the muscle cells must have available to them the nutrient substrates, enzymes, and coenzymes for electron transfer, as well as O_2. The lack of any of these during the energy generating process will result in inability to continue furnishing the fibers with energy except through the more limited and temporary pathway of anaerobiosis. The latter leads to the formation of lactic and pyruvic acid, which must eventually be oxidized or reconverted to glucose or glycogen.

PRINCIPLES OF EXERCISE PHYSIOLOGY

Energy Transfer in Exercise

Dynamic exercise provides the greatest demand for the expenditure of energy by the human body. During such exercise, the ability to produce power through energy transformation represents the most important consideration for physical performance. In events that feature the expression of maximal power, for example, maximal lifting or pushing, the energy output from the exercising muscles may be as much as 120-fold greater than from the muscles at rest, with the energy coming almost entirely from the stored phosphagens. During less intense but sustained exercise, such as long distance running, the energy requirement may be 20- to 30-fold above a resting condition. However, in this situation virtually all of the energy is provided by aerobic metabolic processes, and the power output is in the range of 25% to 30% of maximal potential power. Therefore, the relative contribution of the body's various means for energy transfer can differ markedly, depending on the intensity and duration of exercise, and the power capacity (fitness) of the participant. The stored phosphagens, anaerobic glycolysis, and the aerobic or oxidative system are the energy systems which support the wide range of physical activities and power outputs of which humans are capable.

Immediate Energy: The Phosphagen System

Physical activities of short duration and high intensity, where the power developed by the exercising muscle is near or at maximal level, require an immediate and rapid supply of energy. This energy is provided almost exclusively from the high-energy phosphates, ATP, and CP, stored within specific muscle fibers and activated during the exercise.

Approximately 5 mmol of ATP and 15 mmol of CP are stored within each kilogram of muscle.[4] For a 70-kg person with a muscle mass of 30 kg, this represents between 570 and 690 mmol of high-energy phosphates. If 20 kg of muscle are activated during exercise, then there is sufficient stored phosphate energy to walk briskly for about 1 minute, run a cross-country race for 20 to 30 seconds, or perform sprint activities for about 6 seconds.[5]

All types of physical activity require the utilization of high-energy phosphates, but many, as stated above, rely almost exclusively on those available from muscle stores. For longer duration exercise and for recovery from maximal exertion, additional energy must be generated for the replenishment of ATP.

Short-Term Energy: Anaerobic Glycolysis

For strenuous exercise to continue beyond a brief period of time, high-energy phosphates must be continually resynthesized at a rapid rate. During exercise lasting beyond a few seconds, energy to phosphorylate ADP comes mainly from glucose and stored glycogen during the anaerobic (O_2 lacking) process of glycolysis, with the resulting formation of lactic acid. This allows for the rapid formation of ATP by substrate-level phosphorylation, even though the O_2 supply is inadequate or the energy demands outstrip the capacity for ATP resynthesis aerobically. This anaerobically generated energy for ATP resynthesis can be thought of as reserve fuel that is brought into play during the final seconds of a mile run. In addition, it is of critical importance to supply the rapid energy above that available from stored phosphagens during such events as the 440-yd run or 100-yd swim.

Long-Term Energy: The Aerobic System

Although the energy released in glycolysis is rapid and does not require O_2, relatively little ATP is resynthesized in this manner. Consequently, aerobic reactions provide the important final stage for energy transfer, especially if vigorous exercise exceeds several minutes.

During relatively low intensity, long duration exercise, the amount of O_2 consumed rises rapidly during the first few minutes. By the third or fourth minute, a plateau is reached where the oxygen consumption (VO_2) remains relatively stable for the remainder of the exercise period. This steady rate reflects a balance between the energy required by the exercising muscles and the rate of ATP production through aerobic metabolism. Thus, oxygen-consuming reactions supply the energy for exercise, and any lactic acid produced is either oxidized or reconverted to glucose. Under steady-rate metabolic conditions, lactic acid accumulation is minimal.

The Energy Continuum of Exercise

Figure 13-7 illustrates the relative contribution of the three energy sources during various durations of exercise. The actual contribution that each source makes to total energy provision, however, is difficult to determine. Recently, it has been estimated[6] that the maximal power of the phosphagen

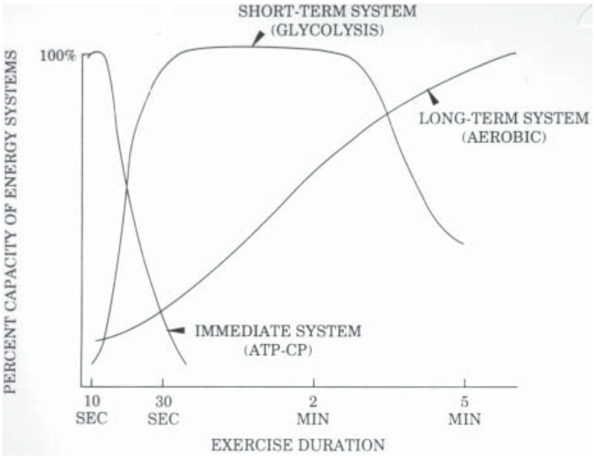

Fig. 13-7. Relative contribution of the three energy sources as a function of exercise duration. Reprinted with permission from McArdle W, Katch F, Katch V. *Exercise Physiology, Energy, Nutrition, and Human Performance.* 3rd ed. Philadelphia, Pa: Lea & Febiger; 1991.

system is reached within 10 seconds, while that of the glycolytic system occurs between 16 to 30 seconds of maximal exercise. Exercise performances of longer duration require an increasing dependence on O_2 to meet the energy requirements of the exercising muscle. As seen in Figure 13-7, approximately 50% of the energy requirement is met by oxidative energy sources by 60 seconds of maximal exercise, while at 120 seconds, this percentage has reached nearly 80%. However, it must be recognized that energy provision for physical exercise is not linked sequentially, and that considerable overlap among the three systems occurs.

Pulmonary Function During Exercise

Dynamic exercise increases the requirement for the utilization of O_2 and the production of carbon dioxide (CO_2) at the cellular level. Thus, the process of pulmonary ventilation must keep pace to allow venous blood to become oxygenated and to maintain arterial CO_2 and hydrogen-ion homeostasis. Indeed, the ventilatory control mechanisms keep arterial CO_2 tension, hydrogen-ion concentration, and O_2 tension remarkably constant despite the marked increase in CO_2 production and O_2 utilization. The exceptions are when exercise is severe enough to produce an elevation in the blood lactic acid concentration, or during exercise at high altitude, or in certain pathophysiologic states.[7]

Regulation of Ventilation During Exercise

The mechanisms involved in the regulation of ventilation are complex and not fully understood. Information is relayed to the medulla through elaborate neural circuits from higher centers in the brain, from the lungs, and from other sensors throughout the body that contribute to the control of ventilation. In addition, the gaseous and chemical states of the blood that reaches the medulla and the chemoreceptors in the aorta and carotid arteries also act to control alveolar ventilation. As a result, relatively constant alveolar gas pressures are maintained even during exhaustive exercise.

Ventilation in Steady-State Exercise. The ventilatory response to constant-intensity exercise is characterized by three phases (Figure 13-8): (1) an abrupt increase in ventilation with the onset of exercise (phase I); (2) a further, more gradual increase to a steady state (phase II); and (3) a steady-state level (phase III). The magnitude of the response in phase I varies with the exercise intensity as well as among individuals. At moderate levels of exercise, this response may account for as much as 50% of the total response (phase III) while at higher exercise intensities, it represents a much smaller fraction of the phase III response.

The increase in ventilation that occurs with moderate exercise is primarily the result of an increase in the depth of respiration. However, as exercise becomes more strenuous, there is an accompanying increase in breathing frequency. At cessation of exercise, there is an abrupt decrease in ventilation, followed by a gradual, exponential decline to preexercise levels.

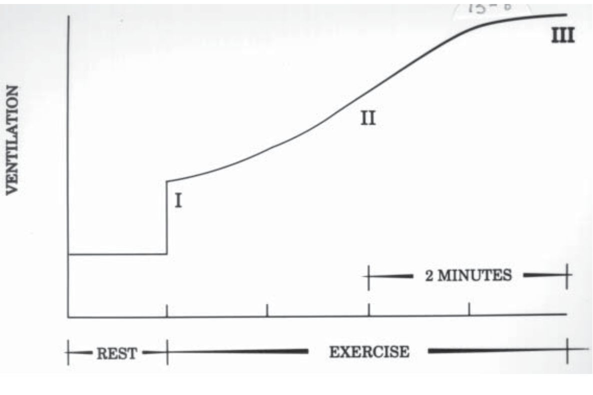

Fig. 13-8. The three phases of the ventilatory response during steady-state exercise.

Control of Ventilation. The rapid response of the ventilatory system at both the onset and cessation of exercise strongly suggests that this portion of exercise hyperventilation is mediated by both cortical and peripheral neurogenic factors. Neural outflow from regions of the motor cortex, as well as cortical activation in anticipation of exercise, stimulates the respiratory neurons in the medulla. In addition, afferent impulses from proprioceptors in muscles, tendons, and joints may influence the ventilatory adjustments to exercise. Although such peripheral receptors have not been identified, experiments involving electrical stimulation of muscles, and voluntary exercise with muscle blood flow occluded, support the existence of such mechanoreceptors in producing a reflex hyperventilation.[8]

The more gradual increase in ventilation during exercise is presumably due to gaseous or chemical humoral factors even though arterial pH, partial pressure of carbon dioxide (PCO_2), and partial pressure of oxygen (PO_2) remain constant during moderate exercise. An increase in body temperature may play a role since it has a direct stimulating effect on the neurons of the respiratory centers in the medulla, and probably exerts some control over ventilation in prolonged exercise. In addition, it may be that the sensitivity of the respiratory center to CO_2 is increased or that the respiratory fluctuations in arterial PCO_2 increase so that, even though the mean arterial PCO_2 does not rise, it is CO_2 that is responsible for the increase in ventilation. Thus, the control of ventilation during moderate exercise is not the result of any single factor but rather the combined, and perhaps simultaneous, result of several humoral and neural stimuli.[9]

Ventilation in Non–Steady-State Exercise. In exercise where the intensity is not constant but increases steadily, the minute ventilation and the rates of VO_2 and CO_2 production increase linearly until a level corresponding to approximately 60% (VO_2 of 2.5 L/min) of the individual's maximal exercise capacity is reached (Figure 13-9). Above this level of exercise intensity, the relationship between minute ventilation and VO_2 becomes curvilinear, increasing disproportionately with the increase in VO_2. As a result, the ventilatory equivalent (ratio of ventilation to VO_2) may increase to 35 or 40 L of air per liter of oxygen consumed. During moderate, steady rate exercise, this ratio is usually maintained at about 25:1.

During moderate, steady-rate exercise, sufficient O_2 is supplied to the exercising muscles. Under these conditions, lactic acid production does not exceed lactic acid uptake and there is no increase in blood concentration of lactic acid. However, as exercise intensity increases, blood lactate begins to increase above baseline levels. The baseline level is termed the *lactate threshold* or point of onset of blood lactate accumulation (OBLA). This normally occurs at an exercise intensity equivalent to 55% to 65% of the VO_2max in healthy, untrained individuals. Almost all of this excess lactic acid is buffered by the sodium bicarbonate system so that hydrogen ion concentration is maintained within acceptable limits. The excess, nonmetabolic CO_2 released by this buffering system stimulates an increase in ventilation and the CO_2 is exhaled into the atmosphere.

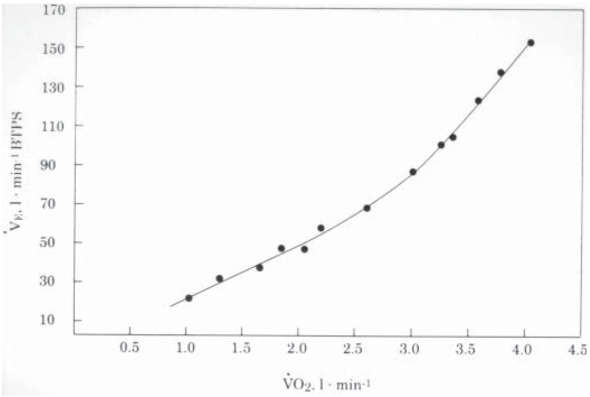

Fig. 13-9. Ventilatory response during non–steady-state exercise.

The exact cause of OBLA is controversial.[10] It has been assumed that OBLA represents an anaerobic state within the muscle. However, it has been shown[10] that muscle lactic acid accumulation is not necessarily linked to muscle anaerobiosis. Lactic acid can accumulate in the presence of adequate muscle oxygenation, implying an imbalance between lactic acid appearance in the blood and its subsequent rate of disappearance. This imbalance may not be only a result of muscle anaerobiosis, but also due to decreased lactic acid clearance, or increased lactic acid production in specific muscle fibers. Thus, caution is urged in interpreting too broadly the specific metabolic significance of OBLA.

Cardiovascular Function During Exercise

In understanding the function of the cardiovascular system during exercise, one must balance the interactions of the heart, blood vessels, and the nervous system. In addition, an integrative approach must be taken to fully appreciate the response by the cardiovascular system to the stimulus of exercise and the adaptations which occur with physical training.

Oxygen Consumption and Cardiac Output

As the intensity of exercise increases, the heart responds by increasing cardiac output (C.O.) in a linear fashion to meet increased metabolic demands. C.O., the product of stroke volume (SV) and heart rate (HR), increases approximately 6 L/min for every 1 L/min increase in VO_2.[11] This relationship is remarkably consistent during all types of exercise. The precise matching of C.O. and VO_2 is brought about by an intricate system of sensory nerves that informs the brain of the body's needs. Indeed, the maximum rate of VO_2max is largely influenced by maximal C.O. Maximum oxygen consumption is defined as:

$$VO_2max = HRmax \bullet SVmax \bullet a\text{-}vO_2diffmax$$

where the latter is the maximal difference between the arterial and mixed venous oxygen content. The $a\text{-}vO_2diffmax$ represents the body's ability to extract and utilize O_2 delivered by the blood to the tissues. With an increase in exercise intensity, more arterial blood is directed to the contracting muscles which extract more O_2; therefore, less oxygen is left in venous blood and the $a\text{-}vO_2diff$ increases.

Heart Rate

The nerves that control HR originate in the medulla. As a person moves from rest to mild exercise, the increase in HR up to approximately 100 beats/min is predominantly caused by suppression of parasympathetic activity, that is, the normally occurring parasympathetic inhibition is removed. Increases beyond 100 beats/min are brought about primarily by stimulation of the sympathetic nerves.[12]

The medulla, as previously seen for ventilation, receives sensory inputs from many areas in control of HR. One of the most important of these areas is the motor cortex. This part of the brain must progressively recruit more muscle fibers as exercise becomes more intense and as previously recruited fibers fatigue. As more fibers are recruited, the motor cortex simultaneously stimulates the medulla to increase HR (as well as ventilation, blood pressure [BP], etc.). The medulla also receives input from nerves in the contracting muscles that are sensitive to changes in the chemical environment and signal a need for increased blood flow. Thus, while the medulla receives many types of inputs that affect the regulation of HR, inputs from the motor cortex and the muscle are among the most important during exercise.

In exercise where a large muscle mass is involved, for example, running, swimming, cross-country skiing, and where the intensity is sufficient to elicit VO_2max, the medulla activates the sympathetic nerves to the heart to increase its rate to maximum. As a rule, HRmax is approximately 220 minus the individual's age in years. The decrease in HRmax with age is caused by a decreased sympathetic drive from the medulla.[13]

Exercise involving a relatively small muscle mass, such as weight lifting with the arms, is unlikely to raise HR much above 150 beats/min, even with maximal power production.[14] This lower maximal HR is a function of the reduced input to the medulla from both the motor cortex and the exercising muscles, so that fewer muscle fibers are being recruited.

HR may be similar during different types of physical activity, in which the VO_2 varies considerably. For example, performing upper body exercise, such as arm curls at an HR of 130 beats/min requires much less VO_2 than does running at the same HR because a much smaller muscle mass is involved in the arm exercise. Thus, when exercise training is prescribed solely on the basis of HR, important exercise effects on the heart may be overlooked.

Arterial Blood Pressure

Information about BP is relayed to the medulla by sensory nerves from baroreceptors in the aorta and carotid arteries. In turn, appropriate adjustments in C.O. and blood vessel diameter are made to maintain BP at a suitable level. BP is the product of C.O. and total peripheral resistance (TPR), the latter being determined primarily by the extent to which small arterial vessels are constricted.

During exercise, such as running, systolic BP increases moderately as the intensity increases, largely because increased C.O. forces more blood into the arterial system, while TPR decreases slightly.[13] On the other hand, during weight lifting, when a small muscle mass contracts very intensely, especially during an isometric contraction, systolic BP can rise to very high levels.[15] This occurs because intensely contracting muscles compress small arteries in those muscles and drive TPR up at the same time that C.O. is increasing.

Diastolic BP does not change much during exercise involving large muscle groups, even when there is an increase in the intensity of the exercise. However, during weight lifting or during isometric contractions with small muscle mass, progressively more small arteries are compressed and even closed completely as contractions become more intense. Thus, during this type of exercise, diastolic BP rises progressively, and TPR is progressively increased, even during cardiac filling.[16]

Blood Flow Distribution

Increased C.O. during exercise must be appropriately distributed to the various regions of the body. This distribution of blood depends on the degree to which the small arteries in the exercising muscles, resting muscles, and other regions of the body are open, and is controlled to a large extent by the medulla and by chemical changes that occur in the contracting muscle. As exercise intensity increases, local vasodilator substances released by the contracting muscle fibers relax the smooth muscle of the arterial walls, causing the arteries to dilate, and allowing more blood to perfuse the muscle.[16] In theory, in heavy exercise involving many muscle groups, local vasodilators could cause arterial dilation to the extent that blood flow approaching 60 L/min would occur.[17] Since maximal C.O. rarely exceeds 35 L/min, even in elite athletes, such degrees of vasodilation would cause a sharp drop in BP reducing blood flow to the brain and heart. However, neural outflow from the medulla, which leads to vasoconstriction of vessels in active muscle and in other regions of the body, can compensate for any excessive local vasodilation,[13] but in so doing, blood flow to the exercising muscle may be limited, or less then metabolic demands require.

As exercise intensity increases, the medulla directs sympathetic vasoconstrictor stimuli to the arteries that supply the stomach, intestines, liver, and kidneys, as well as inactive muscles, reducing blood flow so that a greater fraction of blood is directed to the exercising muscles. Arterial vessels in the skin are also constricted at the onset of exercise, but this effect is ordinarily reversed as the core temperature rises.

With an increasing heat load, temperature control centers in the hypothalamus instruct the medulla to dilate vessels in the skin so that heat can be dissipated to the environment. However, when exercise intensity exceeds 80% to 90% of VO_2max, blood flow to the skin may again be reduced because the temperature regulation needs of the body are overridden by the requirement to maintain BP.[13] In essence, the muscles and the skin compete for blood flow during heavy exercise, and when confronted with this choice, the medulla determines that the muscles win. As a consequence, the reduced skin blood flow can cause marked increases in body temperature.

Stroke Volume

Stroke volume increases from rest to mild exercise (up to approximately 50% VO_2max) and then remains fairly stable with increasing exercise intensity.[13] During exercise involving a small muscle mass, SV is relatively low due to the high TPR that is present. This rise in TPR causes so much resistance to blood flow that SV is reduced; consequently, the flow of blood back to the right side of the heart is lowered. This reduction in venous return to the heart further compromises the ability of the heart to effectively pump blood.

In contrast, exercise with a large muscle mass elicits a larger SV because the relatively low TPR results in a much larger muscle blood flow. Venous return to the heart is enhanced as well, stimulating baroreceptors in the right side of the heart and in the pulmonary circulation.[17] These low pressure receptors send sensory signals to the medulla that cause it to decrease TPR in the exercising muscles, thereby counteracting the increased C.O. to maintain a stable BP simultaneously with a high muscle blood flow and a large venous return to the heart.

TABLE 13-1

ACUTE RESPONSES TO MAXIMAL EXERCISE IN A YOUNG ADULT MAN

Variable	Rest	Exercise	Ratio
O_2 uptake (L/min)	0.30	4.00	13:1
Minute ventilation (L/min)	8	140	18:1
P_{AO_2}	100	115	1.2:1
Pa_{O_2}	95	90	0.95:1
C.O. L/min	6	24	4:1
HR (beats/min)	70	200	2.9:1
Systolic BP (mm Hg)	120	180	1.5:1
Diastolic BP (mm Hg)	80	95	1.2:1
a-v_{O_2} difference (mL/dL)	5	18	3.6:1

C.O.: cardiac output
HR: heart rate
BP: blood pressure
P_{AO_2}: partial pressure of oxygen, alveolar
Pa_{O_2}: partial pressure of oxygen, arterial

Oxygen Transport Variables

Table 13-1 shows commonly found changes in the principal O_2 transport variables that can be measured in healthy volunteers during whole body exercise at intensity levels of VO_2max. These data are round numbers and are not intended to be precisely consistent. There is, of course, considerable variation among individuals in most of these variables. The 13-fold increase in VO_2 from rest to maximal exercise is accomplished by a combination of central and peripheral factors, as previously discussed. The three major responses are in ventilation, C.O., and O_2 extraction by the muscle. Ventilation increases some 20-fold; C.O. and O_2 extraction by about a factor of three to four each. The remaining factors in Table 13-1 change little from rest to maximal exercise.

In summary, all components of the O_2 transport pathway undergo responses to acute exercise. In terms of O_2 supply and thus VO_2max, the changes of significance are in ventilation, blood flow, and muscle tissue O_2 extraction. The resulting VO_2max represents the integrated effects of both central and peripheral factors involved in these three processes.

BODY COMPOSITION AND PHYSICAL FITNESS

Background

The relative composition of the body, particularly in respect to fat and muscle, is often included as one component of physical fitness. Although lying outside our energy pathway definition of physical fitness as presented earlier, the amount and location of both fat and skeletal muscle are strongly associated with physical performance capacity. These components can also be modified through physical training and therefore deserve attention in any discussion of physical fitness, performance, and training.

The term *body composition* refers to the proportional makeup of the body into major components of fat, bone, muscle, and other soft tissues, each of which can be further separated into the chemical constituents. Since body fat, or its excess, is often the focus of interest with respect to physical fitness, the term *fat-free mass* is often used to denote all tissues other than the fat mass. Therefore, in the most basic division, the body is composed of fat and fat-free mass. This simple division is also significant in that the commonly used underwater weighing method to estimate body composition results in two compartments that can be separated by density: fat and fat-free mass. Within the fat-free mass component of healthy individuals, bone, organ, and structural tissues are largely fixed in amount and only skeletal muscle is modifiable. Thus, observed changes in fat-free mass can most often be interpreted as changes in muscle mass.

Body Fat

Body fat can be divided into two categories: (1) essential or obligatory fat, and (2) storage fat. Obligatory fat is lipid, which is required for cell and organ structure, protective cushioning of the organs, nutritional support of specific metabolic activities such as myocardial contractility, and small amounts for cellular functions. Women have additional obligatory fat in the context of sex-specific fat: breast fat and fat associated with the reproductive organs. The amount of the second component, storage fat, is primarily a function of caloric balance, and eth-

TABLE 13-2

REFERENCE BODY COMPOSITION VALUES FOR NONATHLETIC YOUNG ADULTS*

Variable	Women	Men	Ratio (w/m)
Total Fat			
kg (%)	16.8 (28)	13.5 (18)	1.24:1
Obligatory			
kg (%)	7.2 (12)	2.3 (3)	3.13:1
Storage			
kg (%)	9.6 (16)	11.3 (15)	0.85:1
Fat-Free Mass			
kg (%)	43.2 (72)	61.5 (82)	0.70:1
Skeletal Muscle			
kg (%)	21.6 (36)	33.8 (45)	0.64:1
Bone			
kg (%)	7.2 (12)	11.3 (15)	0.64:1
Other			
kg (%)	14.4 (24)	16.4 (22)	0.88:1

*Values represent a compilation of available data.

nic, genetic, and sex-related patterns. Reference values for obligatory and storage fat in nonathletic populations are found in Table 13-2**.**

The regional distribution of storage fat varies between genders and among individuals, and is primarily controlled by the sex hormones and their control of lipoprotein lipase enzyme activity and adipocyte sensitivity to insulin.[18,19] Males predominantly store fat in the abdominal region (android, or central-upper fat pattern) while women typically store fat in the hips and buttocks (gynoid, or peripheral-lower fat pattern).[20] The android pattern reflects greater deep or visceral deposited fat, as opposed to greater subcutaneous fat in the gynoid pattern, and is typically associated with greater muscularity.[21]

Excess storage fat, or obesity, is best expressed in terms of the percentage of body weight composed of fat. McArdle and colleagues[22] have suggested that obesity be defined as 5% units over the population norm. When this is applied to young army members, this equates to 20% (5% above the norm of 15%) for men and 30% (5% above the norm of 25%) for women. These values of 20% and 30% are now employed as the upper allowable limits under the U.S. Army's body weight/fat control program[23] for the age group of 17 to 25 years. In 1974, Behnke and Wilmore[24] supported this when they defined obesity in males as exceeding 20%, since data indicate that fat cells are fully saturated at this level of fatness.

The U.S. Navy, on the other hand, has employed the definition of obesity developed by a National Institutes of Health Consensus Development Conference: "weight for height 20% above the midpoint weight listed in the 1983 Metropolitan Life Insurance tables for the medium frame individual."[25] This value corresponds, in Navy personnel, to 26% body fat in men and 36% in women. Table 13-3 presents a compilation of percentage of body fat values reported for various U.S. military populations.[26-31]

Muscle Mass

The average man has 1.5-fold the amount of skeletal muscle as the average woman, primarily as the result of differences in circulating levels of the male steroid hormone, testosterone. Skeletal muscle represents 45% and 36% of body weight, male and female, respectively (see Table 13-2). Testosterone and other anabolic steroids have potent muscle growth actions, which have led to their use by some body builders and strength athletes.

Overweight, by the customary weight-for-height standards, is usually interpreted as over-fat, although it can actually be a case of being overmuscled. Body builders and athletes requiring high levels of strength or muscular power, or both, typically fall in this latter category. To avoid misclassifying overmuscled individuals as being over-fat, body fat assessment has replaced or supplemented simple body weight measurements in the military services and is discussed later in this chapter.

Physical Training

Changes in body composition, that is, reductions in body fat and increases in muscle mass, are common consequences of physical training, although the extent depends on the nature of the training program, the accompanying nutritional state, as well as the genetic makeup of the individual. Aerobic training, plus caloric restriction, are commonly employed to reduce body fat. Strength or strength endurance (resistance) training, and protein supplementation, are commonly employed to build muscle mass.

Body Fat

Many studies have examined the influence of aerobic exercise training to reduce body fat (eg, Zuti and Golding,[32] and Garrow[33]). Because low to moderate intensities of exercise burn fat almost exclusively, aerobic exercise is an effective means of metabolizing excess fat if, at the same time, caloric intake is controlled. Resistance training is a less efficient means of losing body fat, although reductions of 1% to 3% over a 10- to 20-week program have been shown.[34] Spot reduction of fat from a specific area being exercised is not effective.[35]

Muscle Mass

Resistance training is an effective stimulus to muscle growth, leading to increased muscle mass and cross-sectional area of the trained muscles. Increases of 1.5 kg in fat-free mass over a 10 week period are typical.[34] Training programs employing lower intensities but high volumes (frequencies and durations) are more effective in developing muscle and, therefore, are used by body builders. Some studies[34] show that the increase in muscle size is due to fiber hypertrophy, while others implicate fiber hyperplasia. The evidence for fiber hyperplasia in humans is still controversial and the reader is re-

TABLE 13-3

PERCENT BODY FAT VALUES FROM U.S. MILITARY POPULATIONS AS A FUNCTION OF AGE, GENDER, ETHNICITY, OCCUPATIONAL DEMAND RATING, AND TYPE OF ASSIGNMENT

	Mean + SD	
Category	Women	Men
US Army Recruits (new)[1]		
Age (y)		
17-20	27.7 ± 4.2	15.3 ± 4.7
21-25	28.8 ± 4.5	16.1 ± 5.2
26-30	28.3 ± 4.3	18.1 ± 5.2
30-35	31.0 ± 4.8	22.4 ± 4.6
USMA Cadets[2]	26.5 ± 3.2	12.2 ± 3.0
US Army combat and combat support[3]		
Age (y)		
17-20	26.3 ± 5.3	15.4 ± 5.9
21-27	25.4 ± 6.0	16.7 ± 6.7
29-39	29.2 ± 7.0	21.6 ± 7.0
40+		23.1 ± 5.3
Race		
White	28.0 ± 5.5	17.6 ± 5.5
Black	28.0 ± 4.0	14.0 ± 5.7
Hispanic	28.0 ± 5.0	17.4 ± 6.1
Infantry[4]		
Age (y)		
17-20	—	15.8 ± 4.1
21-25	—	17.9 ± 6.1
26-30	—	19.3 ± 5.9
31-35	—	20.0 ± 5.8
Occupational rating		
Heavy	—	17.2 ± 5.0
Moderate	—	19.6 ± 6.7
Light	—	19.9 ± 6.3
Army Artillery[5]	28.6 ± 3.9	19.3 ± 4.9
Army Special Forces[6]	—	16.1 ± 4.5
Army Ranger students[7]	—	14.6 ± 4.1

Data sources: (1) Knapik JJ, Burse RL, Vogel JA. Height, weight, percent body fat, and indices of adiposity for young men and women entering the U.S. Army. *Aviat Space Environ Med.* 1983;54:223-231. (2) Daniels WL, Kowal DM, Vogel JA, Stauffer RM. Physiological effects of a military training program on male and female cadets. *Aviat Space Environ Med.* 1979;50:562-566. (3) Fitzgerald PI, Vogel JA, Daniels WL, Dziados JE, Teves MA, Mello RP, Reich PJ. *The Body Composition Project: A Summary Report and Descriptive Data*. US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 5-87. (4) Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500. (5) Teves MA, Vogel JA, Carlson DE, Schnakenberg DD. *Body Composition and Muscle Performance Aspects of the 1985 CFFS test.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 12-86. (6) Askew EW, Munro I, Sharp MA, et al. *Nutritional Status and Physical and Mental Performance of Special Operations Soldiers Consuming the Ration, Lightweight or the Meal, Ready-to-Eat Military Field Ration During a 30-day Field Training Exercise.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1987. Technical Report No. 7-87. (7) Unpublished data.

ferred to the review by Saltin and Gollnick[3] for a discussion of the evidence. Growth in muscle tissue may depend on the specific type of training stimulus that is applied. The intensity and volume of training programs can be adjusted to selectively hypertrophy either slow or fast twitch fibers. The fiber hypertrophy is due to an increased size and number of the actin and myosin filaments and additional sarcomeres.[36]

Loss in muscle mass will result from inactivity or severe malnutrition. If the individual is relatively inactive, severe caloric deprivation will result in loss of both body fat and muscle mass.[37] Physical activity during caloric deprivation helps preserve muscle mass so that storage fat is preferentially depleted before muscle mass is used as an energy source.

Body Composition and Physical Capacity

An association between body composition and physical capacity, and performance are readily evident in athletes, as exemplified by the lean, modestly muscled, long distance runner and the heavily muscled, modestly fat Olympic lifter. Whether these relationships, albeit in a less dramatic form, exist in the nonathletic, yet trained population typical of the military services has been the focus of recent studies.[29,38]

Body Fat

Studies by Vogel and colleagues[29] found a correlation coefficient of –0.59 (standard error of the estimate, [SEE] = 4.26) between percent body fat and VO_2max expressed per kg of body weight, for male army recruits beginning basic training and -0.52 (SEE = 5.73) for a diverse sample of soldiers stationed within the continental United States. Similar relationships were found in a larger subsequent study of an army population of 1,117 men and 303 women (Figure 13-10). The correlation coefficient for men was –0.60 (SEE = 5.02) and –0.55 for women (SEE = 3.77). This modest correlation between percentage of body fat and aerobic capacity holds true when aerobic fitness is expressed relative to body weight, but not when capacity is expressed in terms of absolute VO_2max (L/min) as illustrated in Figure 13-11. Absolute aerobic capacity is related to the amount of oxygen-consuming metabolically active muscle, and, therefore, to the fat-free mass, rather than to the metabolically inactive fat tissue. Relative aerobic capacity (VO_2max, mL/min/kg BW) is related to body fat, since increasing fat increases the total body weight (denominator) and thereby lowers the resulting VO_2max value. This corresponds to the physiological situation where the capacity for body propulsion is diminished as body fat adds "dead weight," that is, nonenergy producing mass that must be carried. This is reflected in the association between two-mile-run test scores and body fat content, and is exemplified in long distance runners who typically have the lowest measured body fat content of any athletes, about 4% to 6%.[39]

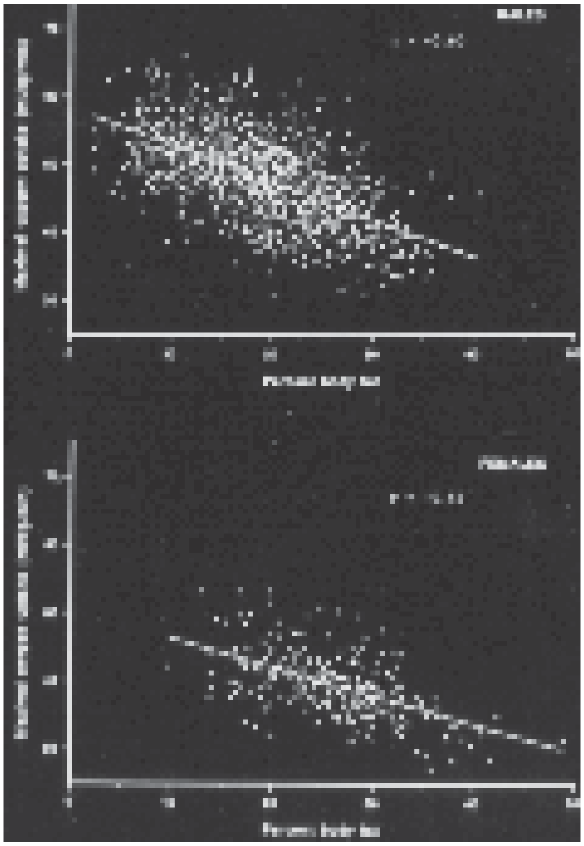

Fig. 13-10. Scatter plot of the relationship between maximal oxygen uptake (per kg body weight) and % body fat in Army men and women. Reprinted with permission from Vogel JA, Friedl KE. Army data: body composition and physical capacity. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy Press; 1992.

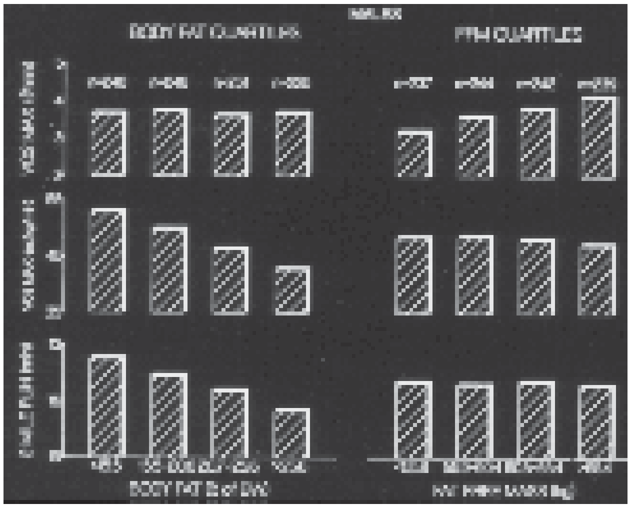

Fig. 13-11. Relation of maximal oxygen uptake to body fat and fat-free mass. Reprinted with permission from Vogel JA, Friedl KE. Army data: body composition and physical capacity. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy Press; 1992.

Muscle Mass

In contrast to aerobic activities that are influenced by percentage of body fat levels, strength activities are related to muscle mass and not related to body fat (see Figure 13-11). Strength of a particular muscle or muscle group is directly related to its cross-sectional area.[40] This relationship holds true for total body muscle mass (as represented by fat-free mass) with total body strength (as represented by

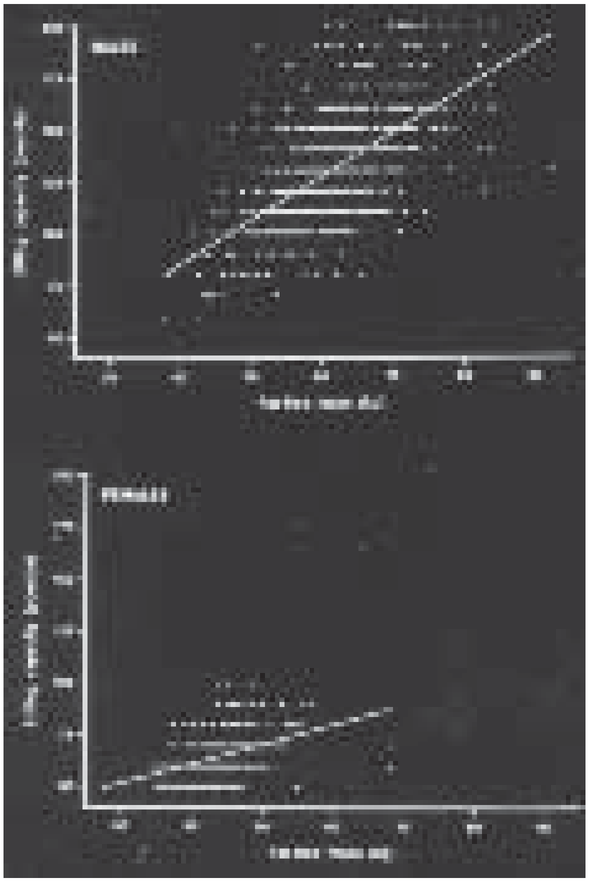

Fig. 13-12. Scatter plot of the relationship between maximal lift strength and fat-free mass. Reprinted with permission from Vogel JA, Friedl KE. Army data: body composition and physical capacity. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance*. Washington, DC: National Academy of Sciences. National Academy Press; 1992:97.

maximal absolute lifting strength) as illustrated in Figure 13-12. Corresponding correlation coefficients are 0.50 (SEE = 20.55) for men and 0.38 (SEE = 11.75) for women.

Body Composition Standards

It was not until 1960 that the U.S. Army first established an upper allowable limit for body weight for entrance into the service, and not until 1976 was a similar standard established for retention (on-the-job). Prior to World War II, the emphasis was on a minimal acceptable weight, and it was not until World War II that this focus changed to overweight.[41] In 1980, the DoD physical fitness directive[1] called for a primary body fat standard rather than just a weight-for-height standard. It also set a DoD goal for percentage of body fat to be 20% for men and 26% for women.

In response to this directive, in 1983 the U.S. Army implemented a body fat standard into its weight control program with a revision to Army Regulation (AR) 600-9, The Army Weight Control Program. This revision retained the weight for height as an initial screen, but employed body fat as the ultimate standard. The standards, as revised in October 1991, are shown in Table 13-4.[24,42-45] This revision also established corresponding body fat

TABLE 13-4

BODY FAT STANDARDS OF THE MILITARY SERVICES (values = body fat as a % of body weight)

US Service Branch	Percentage of Body Fat: Male Accession	Male Retention	Female Accession	Female Retention
Army[1,2]				
Age (y)				
17-20	24	20	30	30
21-27	26	22	32	32
28-39	28	24	34	34
≥ 40	30	26	36	36
Navy[3]				
All ages	—	22*	—	30*
All ages	—	26†	—	36†
Air Force[4]				
Age (y)				
17-29	—	20	—	28
30-39	—	26	—	34
Marine Corps[5]				
All ages	—	18	—	26

*If this value is exceeded, service member is automatically placed on fat loss program.

†If this value is exceeded, administrative action is taken.

Data sources: (1) US Department of the Army. *Medical Service Standards of Fitness.* Washington DC: Department of the Army; 1991. Army Regulation 40-501. 23. (2) US Department of the Army. *The Army Weight Control Program.* Washington DC: Department of the Army; 1991. Army Regulation 600-9. (3) US Department of the Navy. *Physical Readiness Program.* Washington DC: Office of the Chief of Naval Operations; 1986. CNO Instruction 6110.1c. (4) US Department of the Air Force; *The Air Force Weight and Fitness Program.* Washington DC: Department of the Air Force, 1985. Air Force Regulation 35-11. (5) US Marine Corps. *Weight Control and Military Appearance.* Washington DC: Department of the Navy; 1986. Marine Corps Order 6100.1c.

standards for accession into the army, also shown in Table 13-4. Based on patterns of body fat change observed in army recruits,[46] male recruits are allowed to enter the service at four body fat percentage points above their retention standard while females must enter at, or below, their retention standard.

The original body fat standards established by the U.S. Army in 1983 were derived empirically. Working from a base of 20% for the youngest male grouping, 2% was added for each increasing age group, and an 8% increment was added for females for sex-specific fat. The only objectivity in these decisions was the establishment of the base figure of 20%. This value was based, in part, on the finding by Vogel and coworkers[29] that VO_2max began to decline above a body fat of 20%. Continued study of available data has substantiated that the male standards originally established in 1983 are, in fact, supported by both physical performance and appearance criteria.[41] Figure 13-13 illustrates the correspondence between the body fat standard for one age group and corresponding two-mile-run standard. This correspondence for women did not hold up and led to the liberalization of their body fat standards by 2% from the original settings to those now employed as shown in Table 13-4. Thus, the setting of the army's body fat standards are based on physical performance and appearance criteria in agreement with the rationale for the weight control program as stated in AR 600-9, "to ensure that all personnel a) are able to meet the physical demands of their duties under combat conditions, [and] b) present a trim military appearance at all times."[23 (p3)] The history and the basis for the U.S. Army's body weight/body fat standards has been reviewed by Friedl.[41]

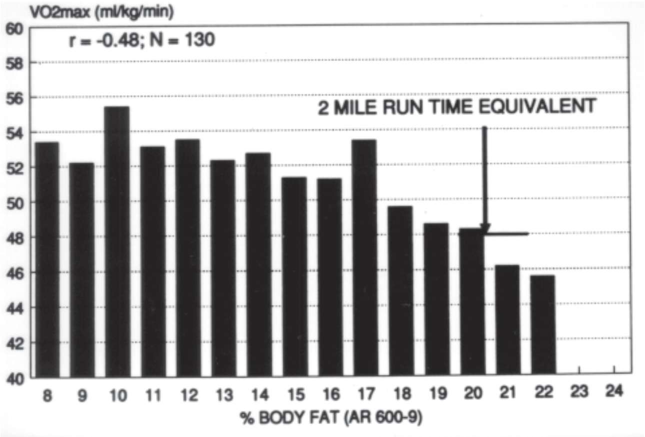

Fig. 13-13. Correspondence between body fat standards (20%) and VO_2max for 17-20 year old male soldiers. Reprinted with permission from: Friedl KE. Body composition and military performance: origins of the army standards. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy of Sciences. National Academy Press; 1992:99.

In contrast to the U.S. Army, the U.S. Navy has based its body fat standards on health criteria because a strong relationship between body fatness and the performance of naval shipboard tasks was not found. Shipboard tasks are predominantly strength-demanding tasks and are unrelated to body fatness. The U.S. Navy moved to adopt a definition of obesity as a weight for height 20% above the midpoint weight listed in the 1983 Metropolitan Life Insurance tables for the medium frame individual, developed from a National Institutes of Health consensus development conference in February 1985. This definition translated into a percentage of body fat for the U.S. Navy population of 22% and 33.5% for males and females, respectively. Adding one standard error to this, the navy established its standard at 26% and 36% for males and females, respectively.[25] Exceeding this limit leads to administrative action, while a more restrictive category, 22% and 30%, requires admission to a fat reduction program. The U.S. Air Force has used appearance as its principal criterion,[44] while the U.S. Marine Corps body fat standards are based primarily on health and appearance requirements.[45]

Body Fat Methodology

Body fat measurement methodology has recently received intense interest and study. The following discussion will be limited to those developments within the military services.

At the time of the DoD directive calling for body fat standards and measurements in the services, most population or "field" measurements for research purposes in the military used the skinfold procedure,[47] based on hydrostatic (underwater) weighing as the criterion method. The hydrostatic procedure employs the known differences in density of fat and nonfat tissue.[48] The skinfold procedure employs a series of skinfold thicknesses, which are summed, and used to predict body fat from empirically derived equations of body density from hydrostatic weighing. Many skinfold prediction equations have been developed, some population specific, and some more generalized.[49] Because most U.S. Army research data on body fat prior to the DoD directive had been collected using the Durnin and Womersley equations,[50] this procedure was adopted for the initial implementation by the army in 1983.

The skinfold procedure suffers from several drawbacks. It can, depending on the equations selected, be population specific since it has been shown that genetic background affects regional fat distribution.[51] The Durnin-Womersley equations were developed from an Anglo-Saxon population and therefore their application to African Americans, Hispanics, and Asians is suspect. Secondly, it is subject to a rather large intermeasurer error, especially outside the research laboratory. Thus the army experienced unsatisfactory variability among body fat measurements throughout many army posts. At the same time, the U.S. Marine Corps had developed simple circumference procedures[52,53] to avoid the problems of measurement variability. Their success inspired the other services to eventually adopt circumference methods for estimating body fat in the "field" for their weight control programs.

The Navy's current circumference procedure and equations were developed by Hodgdon and Beckett[54,55] and are shown in Table 13-5. The army's equations[56] were based on the navy's experience and are similar except for the sites employed for women. The U.S. Air Force has recently adopted the U.S. Navy equations (see Table 13-5). Differences between the equations are the result of different statistical approaches, different limits in what was allowed to enter the regression analysis, and differ-

TABLE 13-5

CIRCUMFERENCE-BASED EQUATIONS EMPLOYED BY THE MILITARY SERVICES TO ESTIMATE PERCENT BODY FAT*

US Service Branch	Gender	R	SEE	Density	Body Fat (%)
Navy and Air Force[1,2]	Male	0.90	3.52	$- 0.191 \bullet \log_{10} (\text{abdom II} - \text{neck}) + 0.155 \bullet \log_{10} (\text{height}) + 1.032)$	$100 \bullet [(3.95/\text{density}) - 4.5]$
	Female	0.85	3.72	$- 0.350 \bullet \log_{10} (\text{abdom I} + \text{hip} + \text{neck}) + 0.221 \bullet \log_{10} (\text{height}) = 1.296$	$100 \bullet [(3.95/\text{density}) - 4.5]$ $100 \bullet [(3.95/\text{density}) - 4.5]$
Army[3]	Male	0.82	4.02		$76.462 \bullet \log_{10} (\text{abdom I} - \text{neck}) - 68.678 \bullet \log_{10} (\text{height}) + 43.742$
	Female	0.82	3.60		$105.3 \bullet \log_{10} (\text{body weight}) - 0.2 \bullet \text{wrist} - 0.533 \bullet \text{neck} - 1.574 \bullet \text{forearm} + 0.173 \bullet \text{hip} - 0.515 \bullet \text{height} - 35.6$
Marine Corps[4]	Male	0.81	3.67		$0.74 \bullet \text{abdom II} - 1.249 \bullet \text{neck} + 40.985$
	Female	0.73	4.11		$1.051 \bullet \text{bicep} - 1.522 \bullet \text{forearm} - 0.879 \bullet \text{neck} + 0.326 \bullet \text{abdom II} + 0.597 \bullet \text{thigh} + 0.707$

*All measurements except height (cm) and weight (kg) are circumferences measured in cm.
R: Correlation coefficient
SEE: Standard error of the estimate
Data sources: (1) Hodgdon JA, Beckett, MB. *Prediction of Percent Body Fat for US Navy Women from Body Circumferences and Height.* San Diego, Calif: Naval Health Research Center; 1984: Report No. 84-29. (2) Hodgdon JA, Beckett, MB. *Prediction of Percent Body Fat for US Navy Men From Body Circumferences and Height.* San Diego, Calif: Naval Health Research Center; 1984: Report No. 84-11. (3) Vogel JA, Kirkpatrick JW, Fitzgerald PI, Hodgdon JA, Harman EA. *Determination of Anthropometry Based Body Fat Equations for the Army's Weight Control Program.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988: Technical Report No. T17-88. Wright HF, Dotson CO, Davis PO. An Investigation of Assessment Techniques for Body Composition of Women Marines. *US Navy Med*. 1980;71:15-26.(4) Wright HF, Dotson CO, Davis PO. An Investigation ofAssessment Techniques for Body Composition of Women Marines. *US Navy Med*. 1980;71:15-26. Wright HF, Dotson CO, Davis PO. Simple Technique for Measurement of Percent Body Fat in Man. *US Navy Med*. 1981;72:23-27.

ent population characteristics. All are based on underwater weighing as the criterion method.

Attention to body composition methodology in the military services is currently causing a reexamination of hydrostatic weighing as the criterion methological standard.[57] Hydrostatic weighing suffers from the fact that it is based on density assumptions that are from a very limited number of cadaver analyses and may not reflect racial and gender differences in bone and nonfat tissue density. The most likely candidate for a replacement of hydrostatic weighing as the criterion standard, and now under study by both the U.S. Army and the U.S. Navy, is dual photon x-ray absorptiometry (DEXA),[58] which employs the differential attenuation of two different energy levels from an x-ray source to separate fat, bone, and fat-free tissue. DEXA can be employed with hydrostatic weighing and body water measurements to derive a four compartment model (fat, fat-free mass, bone, and water) or can be used alone to separate fat and fat-free mass. Initial research[57] supports its potential to replace body density measurements as the sole criterion method.

PHYSICAL CAPACITY FOR EXERCISE

Aerobic Power

Aerobic power, or cardiorespiratory fitness, can be defined as the capacity of the aerobic metabolic pathways to produce energy for moderate intensity exercise for prolonged periods. A highly developed cardiorespiratory endurance capacity is typified by the marathoner, cross-country runner, or cross-country skier. In the military, this component of physical fitness has an important relationship to the performance of such physically demanding tasks as road marching, prolonged load carriage, and the repetitive lifting and carrying of moderate loads. In addition, maintenance of adequate levels of VO$_2$max is important for health protection, body weight control, and the building of unit morale and *esprit de corps*.

Measurement

Exercise scientists agree that the best measure of aerobic power is an individual's VO$_2$max. Indeed, the highest values for VO$_2$max are generally found in elite athletes who compete in such endurance activities as distance running, swimming, bicycling, and cross-country skiing.[59] These individuals have nearly double the aerobic power of age-related sedentary individuals (Figure 13-14). This is not to say, however, that VO$_2$max is a complete expression of aerobic power. Many other factors, especially those at the cellular level, such as number of capillaries, enzymes, and fiber type, exert a strong influence on the capacity to sustain high levels of aerobic exercise.[60] However, the measurement of VO$_2$max provides important information on the overall capac-

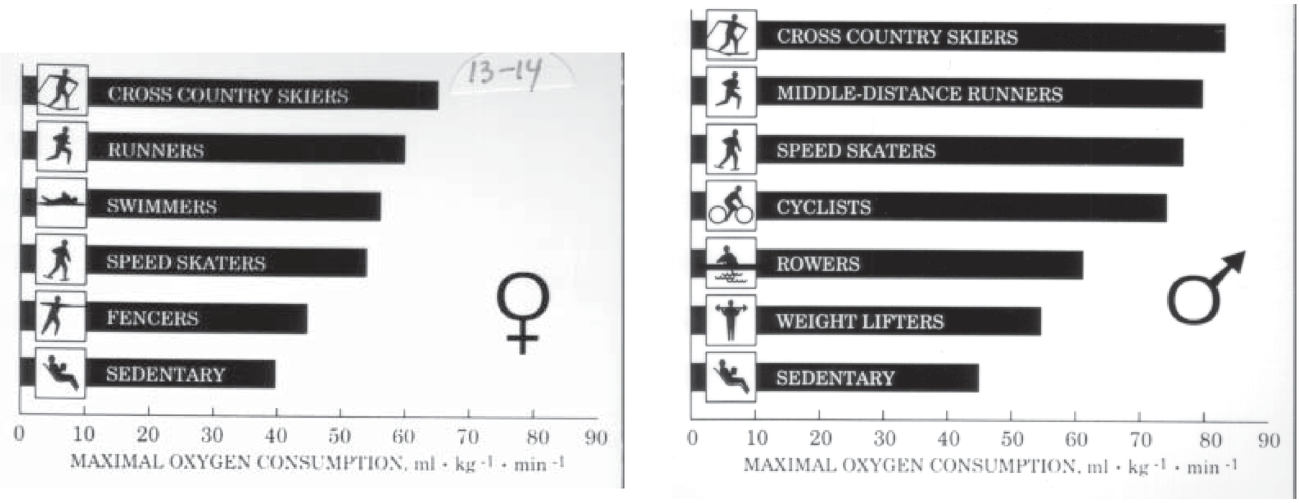

Fig. 13-14. Representative VO$_2$max values of elite athletes. Reprinted with permission from McArdle WD, Katch FI, Katch VL. *Exercise Physiology. Energy, Nutrition, and Human Performance.* 3rd ed. Philadelphia, Pa: Lea & Febiger; 1991.

ity of the aerobic energy system. The VO_2max attainment also requires integration of the ventilatory, cardiovascular and neuromuscular systems which gives VO_2max significant physiologic meaning.[61]

In the research laboratory, and as long as the exercise is of sufficient intensity and duration to permit the maximum transfer of O_2, VO_2max can be determined by various modes of exercise that activate large muscle groups. Typically, VO_2 is measured directly, by the collection and analysis of expired gases while the subject exercises at increasing intensities on either a motor-driven treadmill or cycle ergometer. Although other types of ergometers can be used, these two are by far the most common. As the intensity of exercise increases, the VO_2 increases in direct proportion. Eventually, the individual will reach his maximum ability to deliver O_2 to the exercising muscle, that is, the VO_2 will plateau despite a continued increase in the intensity of exercise (Figure 13-15).

Numerous tests have been devised and standardized for the measurement of VO_2max. In addition to the different modes of exercise, tests that directly measure VO_2 are usually performed either continuously (with no rest between exercise increments), or discontinuously (with the individual resting several minutes between bouts of exercise). In general, the discontinuous method is recommended, if time is available, as it yields slightly higher values, is more comfortable for the subject, and minimizes complications from such factors as local muscle fatigue.

Since the direct measurement of VO_2max requires expensive equipment, trained technicians, and a maximal effort on the part of the participant, investigators have attempted to develop simple, indirect, submaximal tests that would provide accurate predictions of VO_2max and could be administered to large numbers of individuals in a minimum of time and space. Most studies estimating VO_2max from submaximal exercise tests have used the cycle ergometer. The test developed by Astrand and Ryhming,[62] or modifications of this protocol, has been the most extensively used. The basis for the test is the fact that HR and VO_2 are linearly related over a range of exercise intensities. The underlying assumption is that the more fit individual will achieve a higher rate of exercise for the same HR and thus will be able to achieve a much higher exercise intensity before reaching maximum HR.

Determinants

The capacity to generate energy through aerobic metabolism is dependent on the various components of the O_2 transport system as described earlier in the section, Principles of Exercise Physiology. In considering factors that limit this transport, two schools of thought exist, one favoring a central limitation, and the other favoring a peripheral limitation.[63] "Central" O_2 delivery depends on pulmonary ventilation, arterial oxygen content, and C.O. In healthy individuals the first two components do

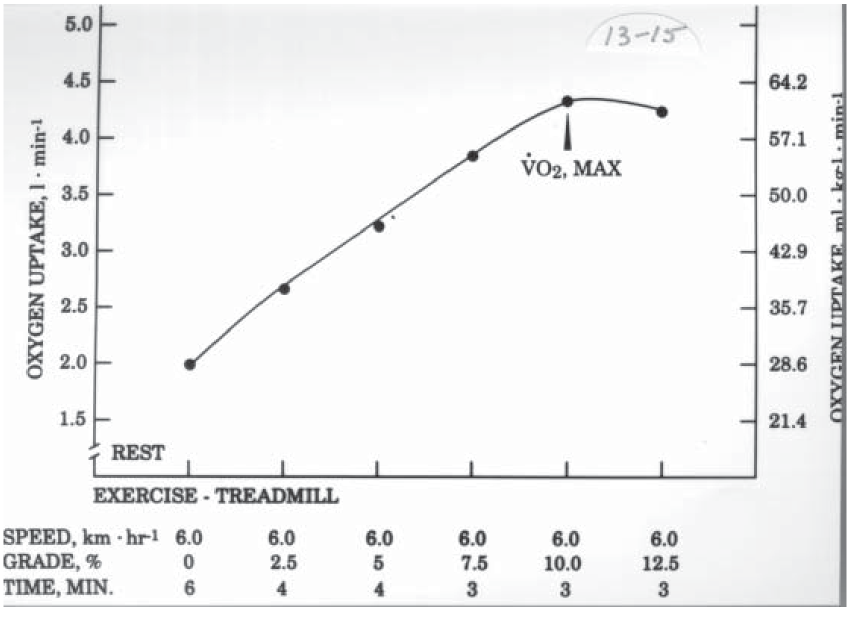

Fig. 13-15. The relation between increased exercise intensity and increased VO_2 and the subsequent determination of VO_2max from the plateau in VO_2 with increased exercise intensity.

not appear to be limiting factors. There is usually considerable reserve in the average person's pulmonary system and, therefore, ventilation does not normally limit exercise performance. In fact, there is no evidence that ventilation reaches a plateau, and it increases exponentially with increasing exercise intensity.

Arterial O_2 content is the product of arterial oxygen saturation and hemoglobin concentration. Neither of these variables changes appreciably from the resting condition up to maximum exercise. Also, in normal, trained, and untrained individuals, there is considerable overlap in hemoglobin and O_2 saturation levels and, therefore, in the magnitude of the arterial O_2 content. This suggests that this component in the O_2 transport system does not limit VO_2max.

Maximum C.O. is determined by the product of maximum SV and maximum HR. Because maximal HR is primarily determined by age and not affected by aerobic fitness level or state of training, the maximum C.O. that can be achieved during exercise is essentially a function of the SV, which becomes, therefore, the prime determinant of the difference in maximum C.O. among individuals. Most studies have also shown a higher SV in maximum exercise as well as submaximum exercise in the trained vs. the untrained individual.

Peripheral factors that may limit the transport of O_2 include local muscle blood flow, extraction of O_2 by the exercising muscle, and the oxidative capacity of the muscle cell. Scientific evidence[13] strongly suggests that muscles are capable of much higher blood flows than they normally receive during maximum exercise, and that O_2 extraction is incomplete even at maximum exercise; both findings imply that the periphery does not limit the maximum transport of O_2. An exception to this may be when limited muscle mass is involved, in which case O_2 extraction may be limiting rather than C.O. The oxidative capacity of the muscle is determined by the number and size of the mitochondria and their concentration of oxidative enzymes. While this could be a rate limiting factor for VO_2max, the evidence[63] suggests that the oxidative capacity of the muscle exceeds the capacity of the circulatory system to transport O_2 to the muscle.

In conclusion, VO_2max depends on the optimal linkage between all components of the O_2 transport system. However, of all the determinants that change with physical training, the cardiac component, that is, C.O., is most adaptable and, within that, it is the SV that is most important.

Anaerobic Power

The term *anaerobic power*, or muscular endurance, refers to the capacity for those types of activities characterized by all-out exercise of brief duration (approximately 5 to 60 seconds), which are fueled mainly by the immediate and short-term (anaerobic) energy systems. In this type of exercise, the energy requirement significantly exceeds the energy generated by oxidation in the respiratory chain. As a result, the anaerobic reactions of glycolysis predominate and are characterized by large quantities of lactic acid accumulating within the muscle cell and ultimately in the blood. Typical military tasks that fall within this category are sprinting, rushing, walking or running with a heavy load, and heavy repetitive lifting.

Measurement

Unlike tests for VO_2max, no specific criteria exist to indicate that a person has reached a maximal anaerobic effort. Indeed, the ability to quantify anaerobic power is difficult due to the considerable overlap that exists in the utilization of the various energy systems during short-term, high-intensity exercise. Efforts to use the measurement of O_2 debt or accumulated lactic acid in the blood have been unsatisfactory in quantifying anaerobic power. Thus, various performance tests have been developed with which to evaluate the capacity for the immediate and short-term means of energy transfer. These tests are generally referred to as power tests, where a maximal force is generated through a distance over a short period of time (approximate range of 5 to 60 seconds).[64]

Maximal sprint runs (time for 50-200 m, or distance covered in a fixed time period) and cycling tests have typically been used to test this energy capacity; although repetitive lifting, shuttle runs, and pushups, situps, or chinups are also tests that have been used. Because of the effects that age, skill, motivation, and body size have on performance, it is difficult to select a suitable criterion test and to develop appropriate norms to evaluate anaerobic power.

Several anaerobic power tests have recently been developed using laboratory ergometers or dynamometers, which provide better precision in quantifying the power output and are more reliable than the performance tests discussed above. One of these is the isokinetic fatigue test, using a Cybex (Lumex Inc.) dynamometer on which the subject performs 50 maximal leg extensions at an angular velocity of

180°/s over a period of 1 minute.[65] Maximal and average torque values are utilized as quantifiable variables. A second test uses a cycle ergometer, where the subject pedals maximally against a very high resistance for 30 seconds.[66] Maximal and average power output values can be calculated.

Determinants

Factors important in an individual's ability to produce high power outputs during short-term exercise are the amount and type of muscle fibers involved in the exercise and the capacity for anaerobic metabolism within those fibers. In tests that measure anaerobic power, there is a strong positive relationship between the amount of power produced and the percentage of fast twitch (type 2) muscle fibers in the exercising muscle. In addition, the greater the muscle mass that is activated during high-intensity exercise, the greater the power output that is achieved.[67] Anaerobic training studies have shown that metabolic changes occurring in the muscle cell include both an increase in the concentration of glycolytic enzymes, and increased levels of anaerobic energy substrates.[68] Thus, the ability to produce power can be attributed primarily to the high anerobic capacity of the type 2 muscle fibers, the percentage of type 2 fibers in the muscle, and the absolute amount of muscle mass available for contraction.

Muscular Strength

The term "muscular strength" is difficult to define in human performance because the term has been employed in various ways by athletes, strength and conditioning specialists, physiologists, physicians, and the lay public. It has been proposed, therefore, that the term "strength" be employed to refer to the maximum force a muscle or muscle group can voluntarily generate at a specified velocity.[69] Physical activities that have a high muscular strength component are those of short duration (less than 5 seconds), where the energy is provided primarily through the splitting of stored phosphagens within the muscle cell, and those that require the generation of high force, for example, the subject must overcome a high resistance. Military tasks that have a large strength component include heavy lifting, pushing, pulling, and throwing.

Measurement

Numerous methods are available to quantify the amount of force that can be generated by a contracting muscle or group of muscles. Which method to use is frequently determined by such factors as availability of equipment, safety concerns of the subject, and the type of muscular contraction to be performed. In general, methods of strength testing are divided into two types: (1) those that measure static or isometric contractions, and (2) those that measure dynamic muscular contractions.

The assessment of static strength requires spring-type or electrical devices that measure the force produced by the muscle and are secured so as to preclude movement. Spring-type devices include dynamometers, spring balances, and the cable tensiometer. Electrical devices primarily include the wire strain gauge connected to a Wheatstone bridge circuit that is permanently mounted to form a load cell.

The cable tensiometer is typically used for measuring muscular force during knee, elbow, and trunk flexion or extension contractions. The tensiometer is lightweight, portable, durable, easy to use, and can be utilized for recording force measurements at various angles in the range-of-motion of a specific joint. Dynamometers, which operate on the principle of compression, have been developed for measuring hand-grip strength, isometric lifting capacity, and upright pull capacity. Load cells are more accurate than the above devices and can be used for assessing the force developed by most any muscle or muscle group.

In contrast to static testing, dynamic strength testing provides for a more functional assessment of strength. Dynamic testing can range from simple tests of the maximum amount of weight lifted in any one movement (eg, elbow flexion or biceps two-arm curl) just one time, to the use of sophisticated laboratory devices that allow the assessment of muscle torque at each point in the range-of-motion. The former, referred to as the one repetition maximum test (1RM), is the maximum amount of weight lifted one time with correct form during the performance of a predetermined weight-lifting exercise, usually using free weights such as barbells. In addition to free weights, there are many types of machines available where movable weights are pushed or pulled through the use of cables or bars. Such machines, however, are more frequently used for training of muscular strength than testing.

The emergence of microprocessor technology has allowed for the development of sophisticated systems that can accurately quantify muscular forces generated during a variety of movements. One such device, the isokinetic dynamometer, allows for the measurement of maximal torque through the full range-of-motion while velocity is held constant.[70]

Determinants

The single most important factor in the ability of muscle to generate force is the total mass of the muscle involved in the contraction. Regardless of gender, human skeletal muscle can generate approximately 3 to 8 kg of force / cm^2 of muscle cross-section. Studies[71] have shown that the greatest force is exerted by individuals with the largest muscle cross-sectional area, and that a linear relationship exists between strength and muscle size. In the body, however, this force-output capacity varies, depending on the arrangement of the bony levers and muscle architecture.

In addition to muscle mass, factors broadly characterized as psychologic or neural can influence the expression of human strength. The initial gains in strength during the first few weeks of a resistance training program without any measurable change in muscle size suggest that such factors are important in muscular force production. These neural factors are related to such processes as increased neural drive to the muscle, increased synchronization of the motor units, increased activation of the contractile apparatus, and inhibition of the protective mechanisms of the muscle (ie, golgi tendon organs), as well as antagonistic muscle.[34] Therefore, the production of maximal force is determined not only by the total number of muscle fibers activated (muscle mass), but also by the coordinated control of motor units.

PHYSICAL CAPACITY OF MILITARY POPULATIONS

A study of military population physical capacity demographics is valuable for several reasons. First, military accessions represent a wide cross-section of the national population and therefore are valuable in assessing national trends and making international comparisons for the young, healthy component of society. Second, population studies within the military can document trends relative to the emphasis and effectiveness of physical training programs, and help establish norms and standards. Third, the data can describe the influence that age, gender, occupation, and unit mission may have on fitness parameters.

The data presented here are largely from the U.S. Army, where most large population studies have been conducted. Where available, both direct laboratory and field tests of fitness are presented.

Aerobic Capacity

Gender

The most prominent factor in the demographics of VO_2max is gender. When factors such as training state and age are taken into account, women have approximately 30% less VO_2max than men. If body weight is taken into account, this difference drops to 25% and, if body fat is taken into account (VO_2max per kg fat-free mass), the difference diminishes to about 12%. The gender difference that remains when body weight and muscle mass are normalized is due to the larger size of the heart, blood and red cell volume, and O_2 transport capacity of the blood of men. These basic physiological differences between genders is recognized by all three U.S. military services in implementing sepa-

TABLE 13-6

EFFECT OF GENDER ON AEROBIC CAPACITY AS ASSESSED BY DIRECTLY MEASURED MAXIMAL OXYGEN UPTAKE (mL/kg BW/min)

	Mean ± SD (n)	
US Army Group	**Women**	**Men**
Army Accessions[1]	37.5 ± 3.7 (212)	51.1 ± 5.1 (210)
Army Trainees[2]	39.2 ± 3.8 (163)	53.6 ± 4.4 (176)
Army Unit Assignees[3]	39.7 ± 4.6 (238)	48.0 ± 6.3 (964)
Army Officer Cadets[4]	49.7 ± 4.2 (26)	60.6 ± 4.7 (29)

Data sources: (1) New recruits prior to basic training, from Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500. (2) Recruits at the completion of 8 weeks of basic initial entry training, from Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500. (3) Soldiers assigned to units within the continental U.S., from Fitzgerald PI, Vogel JA, Daniels WL, Dziados JE, Teves MA, Mello RP, Reich PJ. *The body composition project: a summary report and descriptive data.* US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 5-87. (4) First year cadets at the U.S. Military Academy, West Point, from Daniels WL, Kowal DM, Vogel JA, Stauffer RM. Physiological effects of a military training program on male and female cadets. *Aviat Space Environ Med.* 1979;50:562-566.

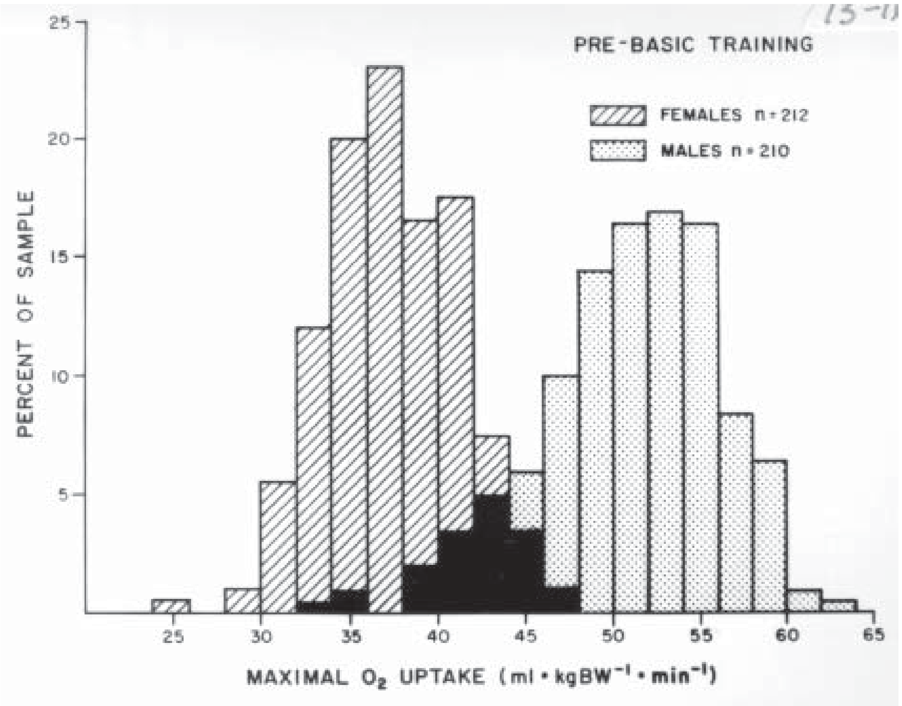

Fig. 13-16. Distribution of Vo_2max in male and female recruits at the beginning of basic training. Reprinted with permission from: Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:497.

rate aerobic fitness standards for the two genders. Available data on gender differences are summarized from several studies in Table 13-6. The minimal overlap between genders in VO_2max (per kg body weight) is illustrated in Figure 13-16. Table 13-7 presents further gender comparisons from estimated aerobic fitness data collected during a large army fitness survey utilizing the two-mile-run event on the physical fitness test.[72]

Age

Age is another generally recognized factor that influences VO_2max. As with gender, the effect of age is, to some degree, unavoidable. Although intense training can reduce or delay the aging effect, its influence can be seen in the majority of the population and, eventually, even in elite athletes.[2] The army takes this into account by adjusting the fitness standards into five age groups. The age effect on aerobic fitness is illustrated in Figure 13-17 and presented in Tables 13-7 and 13-8.

Unit Assignment and Occupation

Other factors that may influence population statistics of aerobic fitness include the type of unit to which the service member is assigned and his occupational specialty. It would be reasonable to ex-

TABLE 13-7

EFFECT OF GENDER AND AGE ON THE US ARMY PHYSICAL FITNESS TEST'S TWO-MILE-RUN FOR TIME FOR THE ESTIMATION OF AEROBIC CAPACITY*

	Minutes:Seconds, Mean (n)	
Age Group (y)	**Women**	**Men**
17-21	18:12 (111)	14:43 (1,157)
22-26	17:43 (228)	14:49 (1,688)
27-31	18:14 (164)	15:09 (1,017)
32-36	19:15 (119)	15:28 (825)
37-41	19:26 (34)	16:01 (382)
42-46	20:08 (18)	16:30 (217)
47-51	21:09 (2)	17:00 (54)

*Standard deviations not available.
Adapted with permission from O'Connor JS, Bahrke MS. 1988 Active army physical fitness survey. *Mil Med.* 1990;12:579-585.

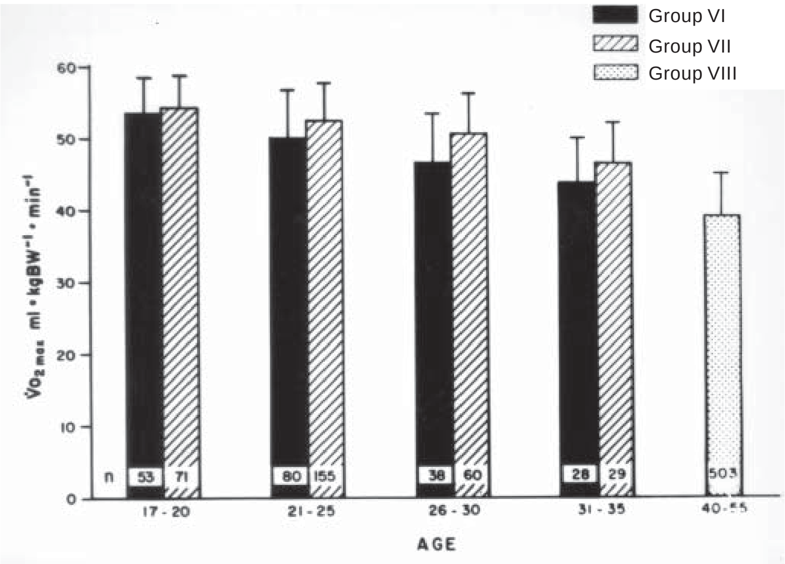

Fig. 13-17. Decline in VO_2max with age in infantry soldiers. Groups representing different training programs: VI—stateside males assigned to units in the Continental United States; VII—males assigned to an overseas combat infantry unit known for its intensive running program; and VIII—males over the age of 40 years. Reprinted with permission from Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:497.

pect that combat units, particularly infantry and infantry occupations, would show among the highest values of aerobic fitness. This may not always be the case because the emphasis placed on physical training by individual and unit training programs can vary greatly and can readily override the occupational effect of the unit or job. Thus, a headquarters or signal unit might show an exceedingly high level of VO_2max because of the commander's emphasis on fitness, regardless of the

TABLE 13-8

EFFECT OF AGE ON AEROBIC CAPACITY, ASSESSED BY DIRECTLY MEASURED MAXIMUM OXYGEN CONSUMPTION (mL/kg BW/min) IN SOLDIERS ASSIGNED TO UNITS WITHIN THE CONTINENTAL UNITED STATES

	Mean ± SD (n)	
Age Group (y)	**Women**	**Men**
17-20	41.2 ± 5.3 (51)	51.9 ± 4.5 (128)
21-27	39.6 ± 3.9 (140)	50.1 ± 5.8 (337)
28-39	38.0 ± 5.4 (46)	45.1 ± 5.7 (276)

Adapted from Fitzgerald PI, Vogel JA, Daniels WL, et al. *The body composition project: a summary report and descriptive data.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1986:Technical Report No. 5-87.

TABLE 13-9

AEROBIC CAPACITY IN A SAMPLE OF US ARMY UNITS*

Unit	n	Mean ± SD
Infantry (Korea)[1]	315	51.9 ± 6.0
Armor (Texas)[2]	62	47.0 ± 5.1
Artillery (Oklahoma)[3]	29	52.1 ± 5.5
Infantry (Washington)[4]	34	53.6 ± 5.6
Infantry (Alaska)[5]	82	57.7 ± 5.2
Infantry (California)[6]	28	58.6 ± 5.7

*Values represent directly measured maximal oxygen uptake during treadmill running (mL / kg BW / min) in male soldiers. Data sources: (1) Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500.73. Knapik J, Daniels W, Murphy M, Fitzgerald P, Drews F, Vogel J. Physiological factors in infantry operations. *Eur J Appl Physiol.* 1990; 60:233-238. (2) Wright JE, Vogel JA, Sampson JB, Knapik JJ, Patton JF, Daniels WL. Effects of travel across time zones (jet-lag) on exercise capacity and performance. *Aviat Space Environ Med.* 1983; 54:132-137. (3) Patton JF, Vogel JA, Damokosh AI, Mello RP, Knapik JJ, Drews FR. *Physical Fitness and Physical Performance During Continuous Field Artillery Operations.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1987. Technical Report No. T 9-87. (4) Knapik J, Daniels W, Murphy M, Fitzgerald P, Drews F, Vogel J. Physiological factors in infantry operations. *Eur J Appl Physiol.* 1990; 60:233-238. (5) Knapik J, Bahrke M, Staab J, Reynolds K, Vogel J, O'Connor J. *Frequency of Loaded Road March Training and Performance on a Loaded Road March.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1990. Technical Report No. T13-90. (6) Mello RP, Damokosh AI, Reynolds KL, Witt CE, Vogel JA. *The Physiological Determinants of Load Bearing Performance at Different March Distances.* Natick, Mass: US Army Research Institute of Environmental Medicine;1988. Technical Report No. T 15-88.

TABLE 13-10

MAXIMAL LIFTING STRENGTH TO 152 CM AS A FUNCTION OF TRAINING STATE USING AN INCREMENTAL DYNAMIC WEIGHT STACK MACHINE (kg)

	Mean ± SD (n)	
Category	**Women**	**Men**
New Accessions[*]	29.5 ± 5.4 (988)	60.6 ± SD (972)
Post Training[†]	34.6 ± 5.8 (495)	65.8 ± 10.8 (476)
Unit Assignees[‡]	30.7 ± 6.8 (302)	61.0 ± 13.6 (1,078)

[*] Prior to basic initial entry training.

[†] Upon completion of basic and occupational training.

[‡] Personnel assigned to various units within the continental U.S.

Adapted from Sharp MA, Vogel JA. Maximal lifting strength in military personnel. In: Kumar S, ed. *Advances in Industrial Ergonomics and Safety IV*. London: Taylor & Francis; 1992.

demands of the occupations or mission. Table 13-9 presents representative values of a variety of units where directly measured VO$_2$max data have been collected. [29,73-77]

Muscular Strength and Muscular Endurance

Population data on strength and muscular endurance in the military services are relatively scarce. A major impediment in gathering such data is the wide variety of methods used and muscle groups assessed. One exception is the measurement of the "one repetition maximal lift" to a standard height as assessed with an incremental lifting procedure and weight stack machine.[78,79] Such a measurement of "whole body strength" has been used extensively in Armed Forces Military Entrance Processing Stations for occupational screening and qualification. Subsequently, this maximal lift measurement has been collected in a number of army studies and has been reported by Sharp and Vogel.[80] Table 13-10 summarizes this measurement as a function of training state. Figure 13-18 contrasts the gender distributions for this measurement. Measured in this way (whole body length), women average approximately one half the lifting strength of men. This is a slightly larger gender difference than that found in the strength of specific muscle groups. Gender differences are typically greater in the upper body and less in the lower body.[81]

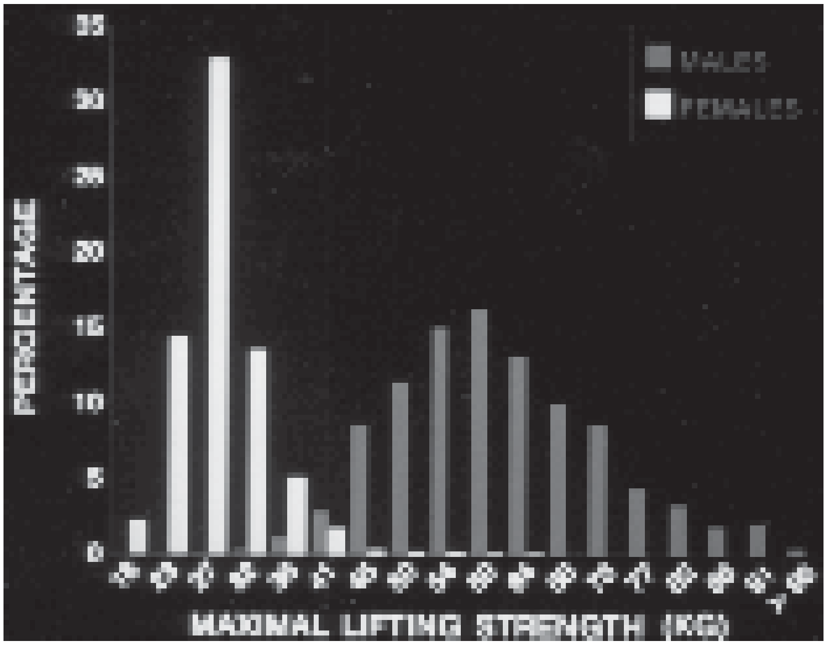

Fig. 13-18. Distribution of maximal lift strength of male and female soldiers. Adapted with permission from Sharp MA, Vogel JA. Maximal lifting strength in military personnel. In: Kumar S, ed. *Advances in Industrial Ergonomics and Safety IV*. London: Taylor & Francis; 1992: 1263.

PRINCIPLES OF PHYSICAL TRAINING

The body's capacity to perform physical activities is a function of its genetically determined body characteristics as well as the extent to which the body and its systems have adapted to a particular type and amount of exercise. While an individual has no control over the genetic component, he can bring about adaptation through an increased level of physical activity. When increased physical activity is formalized into a regular program of scheduled exercises beyond the usual living and occupational activities, it is referred to as *physical training*. Thus, physical training is the activity of imposing increasingly greater loads on the body's systems to bring about adaptive responses that permit the handling of these increased loads with less strain and stress on the individual. Once the desired level of exercise capacity is achieved, physical training must continue to retain the adaptive changes.

The adaptive response appears to have two components; (1) an initial one of depletion or breakdown, which in turn, triggers (2) rebuilding and super repletion, following the pattern of the stress reaction as first proposed in 1957 by Selye.[82] The body's response to physical training follows several well identified principles: overload, progression, specificity, detraining, and overtraining.

Training Principles

Overload

To achieve an adaptation or ability to handle greater amounts of exercise, the body must be challenged with a load greater than that to which it is accustomed. By exercising at a level above "normal" (overload), the body responds physiologically to accommodate this greater load until that load becomes the norm. The overload must be presented progressively and with sufficient intervening recovery time to avoid damage or failure to the systems involved.[83] The added load can be presented by increasing the intensity, duration, or frequency of the training activity. *Intensity* refers to the absolute level of exercise (strength of the stimulus), such as speed of running or the amount of mass lifted. Load can also be modified by adjusting the *duration* of the training bout (minutes that the stimulus is applied) and by the *frequency* (bouts of training per week) of training. Frequently, a combination of intensity, duration, and frequency are used over the course of a formal training program to produce a training overload. Application of these three methods of load adjustment will be discussed at the end of this section.

Progression

It is well established that for training to be most effective, the overload placed on the individual should be given in a regular and gradually increasing fashion. The loading stimulus must be administered frequently enough to retain the adaptation and build on the response. The stimulus then is increased so the new load again becomes an overload. This continues until the desired response is achieved or until no further gains can be elicited. At very high training intensities, large additional loads, usually in terms of duration and frequency, must be added for very small incremental gains. For optimum results, training progression should be individualized, because the starting level and final objective may be quite different among individuals; this, of course, is not usually practical in many military settings. Gains are typically greater at the beginning, more so with the less fit, and then become more gradual with time. Women army recruits typically show large percentage gains in fitness scores during basic initial entry training because of their lower relative level of physical capacity (lower percentage of potential) when they enter the service. Figure 13-19 illustrates the concepts of overload, progression, and recovery during the adaptive process to physical training.

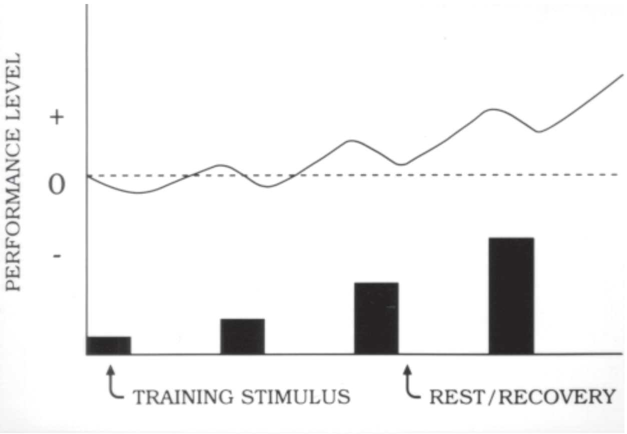

Fig. 13-19. Depiction of the training principles of overload, progression, and recovery.

Specificity

Specificity, in the context of physical training, refers to the concept that adaptive responses to training are specific to the training mode and muscles involved. For example, in general, aerobic training has little effect on muscular strength, while resistance or strength training has minimal effects on VO_2max.[84] Even within the category of aerobic training, run training has the most pronounced effects on running performance and less on other aerobic activities, such as swimming or cycling. This is due to the fact that many training adaptations, as will be discussed later in this section, occur at the cell level of the specific muscles being trained. In resistance training, the responses achieved (strength gained) are generally specific to the speed of contraction, the type of contraction (eg, isometric, isotonic, isokinetic), the muscle group involved, and the energy system utilized (stored vs anaerobic generated energy), although there can be some carry over in strength gained at low isokinetic speeds compared to that measured at higher speeds.

In practice, the principle of specificity should be applied in military physical training programs, in both general physical conditioning and job-specific physical training. For general conditioning, as conducted in basic recruit training, exercises should be selected specific to each of the three categories of fitness. Running is specific to aerobic fitness and will not enhance muscular strength of the upper body. Job-specific physical training should utilize exercises that mimic the anticipated task or that are specific to the muscle groups, intensity, and neuromuscular patterns that occur during the performance of the task; or it should include actual performance of the task.

An example of job-specific physical training would be loaded road marching for the light infantry. While running will provide an adequate base of aerobic fitness, it should not suffice as the only aerobic training for loaded road marching. Knapik and associates[74] found that the addition of road march training twice a month to a well balanced running and resistance training program improved road march performance compared to a program with no road march training. There are many anecdotal reports from military operations in the Falkland Islands and Grenada that load carriage was a major problem in troops who had trained primarily with traditional unloaded running programs.

The movement of artillery projectiles by howitzer crewmen is another task that lends itself to training specificity. Artillery crews should receive a good resistance strength/power program, but it should be supplemented by exercises that simulate the movement patterns employed in the movement of the projectiles.

Detraining

Detraining, or a decrease in the level of exercise capacity, will result when training ceases or when the frequency decreases below a minimum threshold value necessary to maintain the adaptive stimulus for that level of exercise capacity. Thus, once a desired level of exercise capacity is achieved, a level of training must be continued to maintain that level, otherwise it will return to the base level of physical activity; in other words: it is reversible. In the classical bedrest study of Saltin and colleagues,[85] a severe form of detraining, physiological capacity decreased approximately 1% per day. In a more typical form of detraining, Coyle and associates[86] showed that cessation of intense endurance training in track athletes led to a 7% decline in VO_2max over the first 21 days, and after 56 days stabilized 16% lower than initial trained values.

In a review of aerobic detraining research, Neufer[87]suggested that aerobic capacity as measured by VO_2max diminished with detraining, due to a decline in maximal SV and C.O. The latter appears to be related to an initial reduction in blood volume. As the length of detraining continues, O_2 delivery to the tissues will eventually decrease. The specific cause or causes for reduced O_2 delivery are not known.

Less information appears to be available regarding strength and resistance detraining,[88] particularly the time course. Strength detraining leads to muscle atrophy due to diminished fiber cross-sectional area.

Houmard[89] has reviewed the literature concerning detraining on subsequent performance. In well trained endurance athletes, reduction of the weekly volume (frequency and duration) of training by 70% to 80% for up to 3 to 4 weeks will not decrement maximal performance values, as long as training intensity is maintained. Neufer[87] concluded that reductions of 30% to 60% in training volume did not alter aerobic performance as long as the intensity of the exercise session was maintained. This has also been reported for strength training. Furthermore, Coyle and colleagues[86] have shown that athletes who have trained intensely over long periods are more resistant to losses in muscle adaptive changes once training ceases compared to those who have trained only a short period of time. It appears that adaptations to long-term physical training are relatively resistant to change, and major losses can be prevented with modest levels of maintenance training.

Overtraining

Overtraining is a condition characterized by failure to successfully adapt to increased training overload, and is indicated by such symptoms as general fatigue, poor performance, chronic lethargy, and inability to recover from exercise.[90] The probable cause is a too rapid progression in training load, excessive training volume, or inadequate rest and recovery time, or a combination of these. In addition to decrements in exercise capacity, the overtraining syndrome may include a depressed psychological profile, suppressed immune function, and a general disruption of homeostasis.

A state of overtraining is most commonly reported for competitive athletes who are pushing the limits of training tolerance[91]; it is not generally a concern for military unit training. Some possible exceptions include the initial stages of recruit training, elite forces training, and the train-up by individuals attempting to meet a requirement or standard in a short time period, particularly older age service members.

Central to the avoidance of this overtraining syndrome is the gradual application of small increments in loading, combined with adequate rest and recovery. Hard and light intensity workouts, or aerobic and resistance workouts, can be alternated. The recovery period is necessary for homeostasis to be reestablished to the higher level of imposed exercise load. Finally, the signs of overtraining must be heeded. These signs include nervousness or inability to relax, gradual increase in muscle soreness with continued training sessions, unexplained weight loss, inability to complete training sessions, lowered resistance to respiratory infections, loss of appetite, and unexplained drop in physical performance.[88]

The Training Response

Improvement brought about by physical training is the result of adaptive changes that occur in the body that are specific to the type of training. This is a topic of extensive investigation and review.[2,22,34] The primary responses will be reviewed here according to the category of training.

Aerobic Training

Responses to aerobic training occur primarily in the muscle and cardiovascular system. Skeletal muscle responds to aerobic-type training with increased capillarization, vascular conductance, and muscle blood flow during exercise. At the same time, the size and number of muscle cell mitochondria are increased, leading to enhanced levels of oxidative enzymes, thus increasing the oxidative capacity of the muscle. Both this and enhanced muscle blood flow result in greater potential for O_2 delivery and VO_2 by the muscle tissue during exercise.

The primary cardiovascular responses to aerobic training include increased myocardial size and contractility, enhanced blood volume along with a diminished total peripheral vascular resistance. These changes lead to a greater SV, which in turn, permits a greater maximal exercise C.O. The enhanced C.O., combined with the greater O_2 delivery and extraction, results in the greater VO_2max that occurs with aerobic training.

Resistance Training

The most noticeable response to resistance training, as compared to aerobic training, is the enlargement of the muscles being exercised. This enlargement is due to hypertrophy of the individual muscle fibers. This hypertrophy results from an increase in size and number of actin and myosin filaments, and additional sarcomeres within the fibers. Hyperplasia may also occur through the process of fiber splitting, although evidence of this in humans is still debated.

Depending on the nature of the training, the hypertrophy may be selective for fiber type. For example, glycolytic fast twitch fibers show greater enlargement in power lifters who employ high-intensity and low-volume training as opposed to body builders who use lower-intensity and higher-volume training. Increased capillarization of the muscle occurs only in the lower-intensity type of resistance training. Activity levels of the anaerobic enzymes (phosphorylase, phosphofructokinase, hexokinase, lactate dehydrogenase, and pyruvate kinase) change very little with training, and only in the type 2 fibers.

Cardiovascular changes to resistance training are relatively minimal as compared to aerobic training, although some improvement in VO_2max can be achieved. This is particularly true when rest periods are minimized in order to sustain an elevated cardiac stimulus, a prerequisite for improvements in cardiovascular capacity. Cardiac enlargement does occur in resistance training, but for different reasons than in aerobic training. In the latter, the left ventricle enlarges in response to increased demand for SV to supply a greater muscle tissue blood flow demand and a concomitant lower peripheral resistance. Resistance training, on the other hand, causes enlargement because the ventricle must pump against a higher peripheral resistance due to partial occlusion of the vessels during muscle contractions.

In addition to these major changes in the muscle and cardiovascular system, many supportive changes occur with physical training that assist in the adaptation to the higher exercise load. These include changes in the nervous and endocrine systems, connective tissue, and bone.

Neural adaptation plays an important role in resistance training. This includes an increased neural drive to the muscle, increased synchronization of motor units, increased activation of the contractile apparatus, and the release of inhibitory protective mechanisms. Neural mechanisms probably account for the rapid early increase in strength before the slower responding muscle hypertrophy occurs.

The endocrine system also supports the adaptation process in exercise training. The anabolic hormones, testosterone and growth hormone, play a role in the muscle hypertrophy process. Other hormones control the increased substrate availability that is required, that is, insulin, glucagon, thyroxine, and epinephrine.

Finally, structural changes occur in the bone and articular cartilage, which strengthen them to support greater muscle contractile forces. Bone is remodeled when subjected to increased loads with a subsequent increase in bone mineral density and greater mechanical strength. With physical training, articular cartilage increases in thickness, which leads to a greater surface area and compressibility, and both factors will reduce the force per unit area. Training also induces hypertrophy and the strengthening of the tendons and ligaments.[92]

Training Program Components

The effectiveness or gain resulting from a training program depends on the *mode* and extent of the stimulus which is applied. The extent of the training stimulus (or overload) can be further quantified in terms of *intensity* of the workout, *duration* of the exercise period, the *frequency* of the exercise bouts, and the total *length* of the training program.

Mode

The mode of the stimulus refers to the type of physical activity being used, for example, running, cycling, swimming, rowing, and so forth. The mode of activity chosen is important from the standpoint of specificity to the type of gains desired and the effectiveness of the stimulus. Swimming is not an effective training mode for sprinters. Similarly, cycling is not an appropriate training mode for arm and shoulder strength development.

Intensity

Intensity refers to the absolute metabolic or mechanical load imposed by the training program. For aerobic training, intensity is presented typically as a function of velocity, as in running, walking, swimming, skating, and so forth, or grade of incline for walking or running. In stationary cycling, intensity is applied in terms of either mechanical or electrical resistance against pedaling. The intensity of other modes of training, such as climbing, rowing, and bicycling, is more difficult to quantify in absolute terms, and is, therefore, often represented in terms of energy (calories) expended.

In resistance training, intensity is expressed as power output, that is, velocity multiplied by the load or resistance. Thus, intensity of resistance training can be adjusted by changing the speed of the contraction, as well as load displaced or the force exerted against a resistance.

Duration

Duration refers to the time over which the stimulus is applied during a training session and is applicable in both aerobic and resistance training. In resistance training, however, because exercise is typically interspersed with rest periods, it is customary to quantify the "duration" of a resistance training session in terms of the actual number of muscular contractions performed, that is, the number of sets (a period of continuous contractions without rest) and the number of contractions per set performed during a workout.

Frequency

Training frequency refers to the number of training sessions or workouts per week, month, or year.

This is an important descriptive variable since it has been shown that insufficient frequency will not produce gains or maintain an achieved gain, while excessive frequency will lead to overtraining and injuries. The frequency chosen for a particular program will depend on the mode and intensity of training but typically ranges from two to five times per week. This will be further discussed in the subsequent section on training programs.

Length

In physical training, length refers to the time period of a program chosen to meet a desired goal. Typically, programs are 8 to 12 mo long; special applications may be less or much more than this. Training for a particular athletic goal (eg, Olympic competition) may be years in length; training to achieve general fitness and health should be perpetual.

PHYSICAL TRAINING PROGRAMS

Military healthcare providers are consulted about individuals who repeatedly fail periodic physical fitness tests, who are chronically overweight or overfat, who have temporary or permanent medical profiles preventing them from physical training, who are over 40 years of age without medical clearance for testing, and who are new arrivals who may not meet the fitness standards and need individual help. This section is designed to prepare healthcare workers to provide guidance in these situations. Representative examples are given to illustrate the principles and components of training just discussed. The emphasis will be on the individual rather than the unit, although many examples are appropriate to both. Since each fitness component requires separate training activities, each component will be discussed separately, followed by some comments regarding integration into a comprehensive program.

General Considerations

The purpose of any physical training program, whether it be for a unit or an individual, should be to achieve one or more benefits or goals that are identified at the beginning. These goals should not be used for punishment and should not be used simply to pass the periodic physical fitness test. Training programs should have one or more of the following objectives: (*a*) an increase in exercise capacity, which translates into improved physical job performance and physical reserve for emergencies; (*b*) promotion of good health and military appearance (including weight and fat control); and (*c*) promotion of the positive behavioral characteristics of discipline, mental toughness, group cohesion, esprit de corps, and a feeling of self-worth and self-pride. The actual design of individual programs should take into consideration the service member's relative priority among these objectives. Group programs should include all three of these objectives and then vary only in regard to the type and degree of exercise capacity that is to be achieved.

Fitness testing, although not the appropriate end point for a program, is, nevertheless, an important tool in a physical training program. Fitness tests are useful in judging the proper starting point for a program, as well as for measuring its progress. Skilled or experienced individuals can use training activity performance to accomplish the same goal, but prescribed tests are more suitable for the inexperienced or the group training situation.

Finally, several important points of general guidance should be given to the unconditioned individual about to start a training program: (*a*) start slowly, (*b*) progress gradually, and (*c*) incorporate variation. Injuries, discouragement, and loss of interest will occur if the principles of training, particularly those of intensity and progression, are not followed.

Aerobic Training

Mode

An adequate stimulus for the aerobic component of fitness, that is, the various components of the O_2 delivery and utilization system, includes training that involves continuous, rhythmic exercise activities of a large muscle mass. The large muscle mass is needed to create the metabolic demand necessary to bring about the adaptive changes to the heart and circulation. For this reason biceps curls would not be suggested as an aerobic training activity. Appropriate aerobic activities include walking, running, cycling, swimming, rowing, stair climbing, and skiing, or any other rhythmic activity that employs the large muscle groups of the legs, arms, and shoulders. Walking with loads (backpacking) is an excellent aerobic training activity that has the advantage of lower impact forces on the lower extremities than are associated with running.

Frequency

There is ample evidence that a minimum frequency of twice per week, and more preferably three times per week, is necessary to achieve gains and to maintain a desired aerobic fitness goal. Greater gains can be achieved by training up to five times weekly, but this must be weighed against time requirements for other training activities and the potential cost in increased injuries.[93]

Intensity

An adequate stimulus for increasing aerobic capacity is an exercise intensity equivalent to 50% to 85% of VO_2max. More practically, this is equivalent to an intensity level of 60% to 90% of maximal HR reserve (age-adjusted maximal HR minus resting HR). For the beginner or low-fit individual, programs should start at the low end of this range, while those with high initial capacities will need to train at the high end of this range, referred to as the target HR.

Duration

The duration of the aerobic training session is as important as the intensity and frequency. To be effective, the stimulus to the circulation and muscle must be maintained for a minimum of 15 minutes. Training durations of 30 to 60 minutes are optimal, again depending on the starting level and the desired goal.

Summary

Running and loaded road-marching are excellent aerobic activities that take little or no equipment or facilities. Depending on the availability of facilities, other activities can be added or substituted twice a week for 15 to 20 minutes for each session, provided they involve rhythmic activity of large muscle groups at an intensity of at least 60% of maximal HR reserve. Higher intensities, longer training sessions, and more frequent sessions will bring about greater improvement. For more detailed information, the reader is referred to textbooks of exercise physiology and aerobic fitness.[2,23,94,95]

Anaerobic Power Training

Mode

Anaerobic power (muscular endurance) training modes can take several forms: (*a*) traditional resistance exercise with free weights or resistance equipment; (*b*) use of body weight for resistance; or (*c*) adapting traditional aerobic training modes by performing them in a repeated, brief, and relatively intense fashion. The choice between using free weights, weight stack machines, or other resistance machines often depends more on availability than the theoretical advantages of one system or machine over another. The theoretical advantages are more important to the serious competitor and are not of particular concern to the average service member. If no equipment is available, as may be the case in a military unit training environment, an individual can use his own body weight or the weight of a partner for resistance exercise. Many examples of partner-resisted exercises are given in the army's Physical Fitness Training manual.[96] Pushups, pullups, and situps are examples of muscular endurance training using an individual's own body weight.

Common aerobic training modes can be adapted for muscular endurance training by performing them in an anaerobic manner, including sprint running and sprint cycling for the lower body, and sprint rowing for the upper body. Sprint training should employ repeated maximal efforts of 15 to 60 seconds in duration with minimal intervening rest.

Intensity and Duration

Intensity and workout duration are closely related in anaerobic power training. There is a wide range of choices, depending on the specific goal, but the traditional approach employs one-set routines to muscle failure at a relatively low resistance (intensity), for example, 20% to 40% of 1RM. A set refers to a series of movement repetitions without stopping. The 1RM is defined as the maximal amount of weight or resistance that can be moved once. In this case, an exercise training session for a particular muscle group would consist of repeating the movement without rest at 20% to 40% of the 1RM resistance until failure.

Summary

Anaerobic power training programs are quite varied, depending on facilities available, muscle groups chosen, and specific goals. One-set, 20% to 40% of 1RM, is typically used with free weights or other weight and resistance equipment. Other modes of rhythmic exercise can be used if intensity is near maximum and rest periods are minimized. The reader is referred to Fleck and Kraemer,[34] and

O'Shea[97] for further details and examples of muscular endurance training.

Strength Training

Mode

The same training modes mentioned for muscular endurance training can also be used for strength training, but the most effective are free weights or resistance equipment. This is due to the fact that the very high loads (resistance) needed for a strength training stimulus are most readily presented with external weights rather than using body weight, running, or sprint activities. Resistance can be applied in a variety of ways: (*a*) isometrically—constant tension with no range-of-motion activity, (*b*) dynamic constant resistance (also referred to as isotonic)—constant tension throughout the range-of-motion as in typical weight lifting, (*c*) eccentrically—muscle lengthening movement (also called negative resistance), (*d*) variable resistance—changing resistance through the range-of-motion, and (*e*) isokinetic—variable resistance at constant velocity through the range-of-motion. The latter two forms of training require machines that use either cams, lever arms, pulleys, or hydraulics. The choice between these various methods is important only to the serious strength trainer or athlete. The beginner or novice should utilize either dynamic constant resistance or variable resistance, or both, depending on the availability of equipment.

Frequency

The beginner or unconditioned individual should not strength-train more than three times per week. Recovery time is particularly important at the beginning of a strength-training program.

Intensity and Duration

In contrast to muscular endurance training where many repetitions at a relatively low power intensity are employed, strength training optimally requires high intensity with few repetitions between rest. The traditional, time-proven program is performing a training exercise of (*a*) a load of 2 to 6 repetition maximums (the maximum weight or resistance that can be moved in no more than 2 to 6 repetitions), which is equivalent to 85% to 95% of 1RM; (*b*) 3 to 6 sets (one set equals 2 to 6 repetitions); and (*c*) 2 to 3 minutes of rest between sets. Beginners should exercise small muscle groups first and then larger groups, or alternate between upper body and lower body.

This is not to say that low resistance and high repetitions will not produce strength gains as long as the exercise is performed to fatigue. In fact, with an injured individual, to avoid further injuries, it may be preferable to use the less optimal but safer procedure of low resistance and high repetitions.

Summary

The most effective stimulus for strength training utilizes relatively few repetitions between ample rest periods, at near maximal strength intensities. Constant and variable resistance methods are both effective for the beginning and average weight trainer. The reader is referred to Fleck and Kraemer,[34] O'Shea,[97] and Baechle and Groves[98] for additional information.

Training Program Integration

The objective of most military physical training programs is to achieve and maintain fitness in all three categories of exercise capacity: aerobic, strength, and muscular endurance. The next step is to develop a mix of activities during the week to train all three categories. Unit programs may also wish to integrate exercises for flexibility, motor coordination, and sport competition. Table 13-11 gives

TABLE 13-11

EXAMPLE OF AN INTEGRATED TRAINING PROGRAM FOR ALL THREE CATEGORIES OF PHYSICAL FITNESS

Weekday	Category	Activity
Monday	Aerobic	Run 3–6 miles
	Anaerobic	Interval sprint runs (6-8 x 100 m)
Tuesday	Strength	2–3 sets, 8–12 repetitions
	Anaerobic	Pushups, situps, chinups
Wednesday	Aerobic	Run 3–6 miles
	Anaerobic	Circuit weight training, 1 set of low weight, high repetitions
Thursday	Strength	2–3 sets, 8–12 repetitions
Friday	Aerobic	Run 3–6 miles or specialty training, eg, loaded road march

an example program which can be used for unit training or as a guide for individual training. The example is illustrative of how all three categories of training can be integrated during a week's schedule.

Occupational or Mission-Specific Physical Training

The role and importance of training specificity has been pointed out in an earlier section. Running will not completely prepare soldiers for loaded road-marching, and resistance training on a universal gym will not optimally prepare cannoneers to move and load 45-kg projectiles for 155-mm howitzers. For these and other similar physically demanding job tasks, time should be allotted on a weekly or bimonthly basis for additional specificity training.

MEDICAL PROBLEMS ASSOCIATED WITH PHYSICAL TRAINING AND EXERCISE

The benefits and positive gains from physical training are not without costs. These costs are in the form of injury or illness resulting directly or indirectly from the training activity. The importance of this problem, and its cost to the individual in terms of lost time from work or training, as well as the costs of medical care and rehabilitation, have gained attention in the specialties of sports and occupational medicine. The specialties deal with the epidemiology, diagnosis, treatment, and prevention of injuries and illnesses associated with physical activity and occupational work. Concern within the military about the cost of activity related injuries, particularly those associated with physical training, has received considerable attention recently, and active programs in injury epidemiology research are now in progress.[99]

This section will address injuries and illnesses that are associated with physical training and operational (or occupational) activities in the military setting. This will include musculoskeletal injuries, hematological disorders, and cardiovascular disease associated with exercise, and injuries related to exercise in environmental extremes.

Musculoskeletal Injuries

Incidence Rates

Several recent studies[100-102] have documented the incidence of musculoskeletal injuries in army basic initial entry training, and infantry basic and advanced training (one station unit training) and the data are summarized in Tables 13-12 through 13-17. An injury is defined as a complaint leading to a sick call visit for treatment. Table 13-12 presents data for the 8-week army basic recruit training and demonstrates the overall high incidence rates with initial military training. Doubling these monthly rates gives a total percentage incidence of 50% and 27% for women and men, respectively, over the total period of the recruit training. These are similar to the rates reported by Kowal[103] (62% for women, 26% for men) and Bensel and Kish[104] (42% for women, 23% for men). Table 13-12 also shows that the majority of injuries are in the lower extremities. Stress fractures are singled out because of their large cost in limited duty time. The 2-fold higher rate of injuries in women is of particular concern as they move into more physically demanding jobs. Table 13-13 presents incidence rates on the sites and types of injury found during the 12 weeks of basic and advanced (one station unit) infantry training.

One might predict that incidence rates in initial training would be higher than in operational units, but this does not appear to be the case, at least in combat arms units. Recent studies[105] found similar incidence rates in men, about 12% (see Table 13-14).

TABLE 13-12

INCIDENCE OF MUSCULOSKELETAL INJURIES IN EIGHT WEEKS OF US ARMY BASIC COMBAT TRAINING*

Category	Women (n=186)	Men (n=124)	Risk Ratio†
All injuries	50.5%	27.4%	1.8:1
Lower extremity injuries	44.6%	20.9%	2.1:1
Stress fractures	12.3%	2.4%	5.1:1
Time-loss injuries	30.1%	20.2%	1.5:1
Limited duty days (n/100/mo)	129 d	40 d	3.2:1

*Fort Jackson, South Carolina, 1984

†Risk Ratio = female ÷ male injury %

Adapted from Jones BH, Manikowski RM, Harris JM, et al. *Incidence of and Risk Factors for Injury and Illness Among Male and Female Army Basic Trainees.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988. Technical Report No. T19-88.

TABLE 13-13

CUMULATIVE INCIDENCE OF TRAINING INJURIES DURING ONE INFANTRY UNIT'S 12-WEEK TRAINING SESSION (basic plus advanced, 303 men)

Training Injury	Incidence (%)
Injury Site	
Foot	10.9
Ankle	10.9
Knee	10.2
Calf	8.6
Lower back	5.9
Thigh	1.7
Hip	0.3
Injury Type	
Nonspecific pain syndrome	23.8
Muscle strain	8.6
Ankle sprain	6.3
Other sprain	1.0
Overuse knee injury	5.9
Bone stress fracture	3.0
Bone stress reaction	2.0
Fascitis	2.3
Achilles tendinitis	1.0
Bursitis	0.7
Fracture	0.7
Unknown	1.7

Adapted from Cowan D, Jones B, Tomlinson JP, Robinson J, Polly D, Frykman P, Reynolds K. *The Epidemiology of Physical Training Injuries in US Army Infantry Trainees: Methodology, Population, and Risk Factors.* Natick, Mass: US Army Research Institute of Environmental Medicine; Technical Report No. T4-89, 1989.

TABLE 13-14

INCIDENCE OF MUSCULOSKELETAL INJURIES IN MALE ARMY LIGHT INFANTRY SOLDIERS[*]

Category	6th Infantry Div[†] (n = 561)	10th Mountain Div[‡] (n = 351)
All injuries	11.9	27.6
Lower extremity injuries	10.6	10.8
Stress fractures	0.5	0.8
Limited duty rate (n/100/month)	80	114

[*] Rate expressed as % of soldiers per month; based on 2-3 month period.

[†] Fort Richardson, Alaska

[‡] Fort Drum, New York

Data source: Reynolds K, Pollard J, Cunero J, Knapik J, Jones B. Frequency of training and past injuries as risk factors for injuries in infantry soldiers. *Med Sci Sport Exerc.* 1991;23:S40.

TABLE 13-15

COMPARISON OF INJURY AND ILLNESS[*] RATES AMONG US ARMY BASIC TRAINEES (sick call visits/100 soldiers/mo)

Category	Injury	Illness	Risk Ratio[†]
One or more visits			
Women	25.2	24.2	1.0:1
Men	13.7	17.7	0.8:1
Total visits			
Women	39.6	37.2	1.1:1
Men	22.1	26.4	0.8:1
Limited duty days			
Women	129 d	6 d	22.0:1
Men	40 d	8 d	5.0:1

[*] All medical complaints other than musculskeletal injuries.

[†] Risk Ratio = injury ÷ illness rate

Data source: Jones B, Manikowski RM, Harris JM, Dziados J, Norton S, Ewart T, Vogel JA. *Incidence of and Risk Factors for Injury and Illness Among Male and Female Army Basic Trainees.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988. Technical Report No. T19-88.

One notable exception was the lower rate of stress fractures compared to new trainees, apparently a condition primarily found in new recruits and discussed later in this section.

The contrast between these injury rates and those for illness are noteworthy. Table 13-15 makes this contrast for U.S. Army basic trainees and Table 13-16 for infantry trainees and soldiers. While rates were similar in basic trainees, the number of days of limited duty were considerably greater for injuries, particularly for women. In infantry trainees and soldiers, the musculoskeletal injury rate ex-

TABLE 13-16

COMPARISON OF INJURY AND ILLNESS* RATES AMONG ARMY INFANTRY TRAINEES AND SOLDIERS (sick call visits/100 soldiers/mo)

Category	Injury	Illness	Risk Ratio†
One or more visits			
Trainees[1]	14.2	4.5	3.2:1
Soldiers[2]	12.8	10.0	1.3:1
Total visits			
Trainees	34.3	27.0	1.3:1
Soldiers	19.6	12.0	1.6:1
Limited duty days			
Trainees	93 d	18 d	5.2:1
Soldiers	113 d	11 d	11.0:1

*All medical complaints other than musculskeletal injuries.
†Risk Ratio = injury ÷ illness rate
Data source: (1) Cowan D, Jones B, Tomlinson JP, Robinson J, Polly D, Frykman P, Reynolds K. *The Epidemiology of Physical Training Injuries in US Army Infantry Trainees: Methodology, Population, and Risk Factors.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1989. Technical Report No. T4-89. (2) Unpublished data.

TABLE 13-17

COMPARISON OF NUMBER OF DAYS OF LIMITED DUTY DUE TO STRESS FRACTURES AND ILLNESS (limited duty days/100 soldiers/mo)

Group	Stress Fracture	Illness	Risk Ratio*
Basic training[1]			
Women	39	6	6.5:1
Men	8	8	1.0:1
Infantry training[2]	19	18	1.0:1
Infantry[3]	23	11	2.1:1

* Risk Ratio = stress fracture ÷ illness number
Data sources: (1) Jones B, Manikowski RM, Harris JM, Dziados J, Norton S, Ewart T, Vogel JA. *Incidence of and Risk Factors for Injury and Illness Among Male and Female Army Basic Trainees.* Natick, Mass: US Army Research Institute of Environmental Medicine; Technical Report No. T19-88, 1988. (2) Cowan D, Jones B, Tomlinson JP, Robinson J, Polly D, Frykman P, Reynolds K. *The Epidemiology of Physical Training Injuries in US Army Infantry Trainees: Methodology, Population, and Risk Factors.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1989. Technical Report No. T4-89. (3) Reynolds K, Pollard J, Cunero J, Knapik J, Jones B. Frequency of training and past injuries as risk factors for injuries in infantry soldiers. *Med Sci Sport Exerc.* 1991;23:S40.

ceeded the rate of illness (medical complaints other than musculoskeletal), and again, injuries produced considerably more days of limited duty than did illness. These numbers can also be contrasted for just one injury, stress fractures, as illustrated in Table 13-17. Thus, during peacetime, musculoskeletal injuries are a significantly greater cause of lost duty time than all combined illnesses.

Risk Factors of Injury

The first step toward reducing injury rates is the identification of those factors that place individuals at risk. A list of such potential risk factors follows:[106]

- Extrinsic risk factors
  - Training program parameters
  - Footwear
  - Training surface
- Intrinsic risk factors
  - Level of physical fitness
  - Prior level of physical activity
  - Body fatness
  - Gender
  - Age
  - Prior injury history
  - Flexibility
  - Arch height
  - Other anatomical factors

Gender. It has already been pointed out (see Table 13-12) that femaleness is a risk factor for injuries in the army.[100,102] The reasons for this are not clear, but lower prior physical activity and lower fitness levels, particularly muscle strength, would seem to be implicated. Whether women are more prone to report to sick call for musculoskeletal symptoms has not been studied.

Age. The limited data available on age suggest that there is a bimodal relation in injury rate, with higher rates found in the youngest and oldest age groups, and the highest incidence at the oldest age group.[100]

Training program. Two training parameters have been examined systematically in army basic training populations by Jones and colleagues[102]: (1) running vs marching volume and (2) periods of reduced intensity. In comparing two training companies, they found that musculoskeletal injuries were one-fourth less in the company that ran 60 miles during the 12-week training cycle as compared to the company running the more typical 120 miles. Injury incidence appeared to be closely related to cumulative running mileage. In another study, reduced volume of running during the second, third, or fourth week of basic training did not reduce the incidence of stress fractures or total injuries. This period had been hypothesized as a period of increased risk due to the active cortical bone remodeling that would be taking place.

Prior physical activity and fitness level. Jones and colleagues[100] have shown that low self-assessed physical activity level and running volume prior to entering the service were negatively related to risk of injury. The quartile having the slowest one-mile-run time at the beginning of basic training had an injury incidence 3-fold greater than the quartile with the fastest run time.

Flexibility. Development and maintenance of flexibility is commonly promoted in lay literature as a preventive measure against injuries in runners and other sports. However, Jones and colleagues[100] found that the relationship between injury rate and flexibility (toe touch test) is bimodal, with the highest rates in those highly flexible and those least flexible. Moderate flexibility was associated with the least injuries.

Arch height. Another unexpected finding by Jones[102] was the relationship between arch height and injury rate. They found that the lowest injury rate (all injury types) was in the group with the flattest arches and the highest rate in those with the highest arches.

Footwear. A systematic comparison of various military footwear on injury rate has not been made. Combat boots have been implicated as a cause of stress fractures and other overuse injuries, particularly in new recruits. This has led to the extensive use of soft running shoes in aerobic training. The addition of cushioning material to the combat boot has not proven beneficial.[107]

Other possible risk factors. The research by Jones and colleagues[100,102] has also implicated other factors as being associated with higher rates of musculoskeletal injuries: higher body fat (in men), smoking, smaller stature (in women), and bowed legs.

Bone Stress Fractures

Bone stress fractures (or reactions) deserve special attention in any discussion of military musculoskeletal training injuries because of their high cost in terms of lost training time (the cause of the greatest loss of recruit training time), particularly when it occurs during initial entry training. Stress *fracture* is a misleading term since actual fracture lines are often absent. The term "stress reaction" is more suitable in describing the condition of the bone's response to acute overloading through a remodeling process that forms new periosteal bone.

The epidemiology and etiology of bone stress reactions have recently been reviewed by Jones and colleagues.[108] They reported that the incidence of bone stress reactions in military trainees is from 1% to 3% in men and 10% to 20% in women, depending on the specific criterion used. Considerable recent attention has been given to stress fractures by the Israeli Defense Force who have reported a trainee incidence rate as high as 31%,[109,110] although other reports from Israel report lower rates of about 5%.[111] The differences in rates appear to be due to the use of the more sensitive technique of radioactive bone scans. A 1993 work by Scully and colleagues[112] suggests that a high percentage of recruits show stress-positive bone scans without any symptoms.

Epidemiology studies have shown that new trainees are at a much higher risk than "seasoned" troops in whom stress reactions are seldom observed. It is also apparent that women are at a much higher risk than men, even when other risk factors are taken into account, possibly due to differences in bone density. Other risk factors include age, race, and prior level of physical activity or level of fitness.

Brudvig and associates,[113] and Gardner and colleagues[107] have shown a relationship between increasing age and incidence of stress fractures. This may be confounded with decreasing fitness with age, although lower prior fitness and activity may be an independent factor.[107] African Americans are generally reported to have lower stress fracture rates than Caucasians, which is attributed to higher bone densities in African Americans.[114]

The anatomical location of stress reactions or fractures and the timing of their occurrence probably depends on the nature, progression, and severity of training. During World War II, stress fractures were most commonly reported[113] in feet, while contemporary reports most often list the legs. Currently, the peak incidence of stress reactions in military recruits is during the first 2 to 4 weeks, probably depending, to a large degree, on the amount of running included in the training program.

The etiology of stress reactions and fractures is now thought to be the bone's physiological response to overload. As the bone is subjected to acute increases of physical stress, as in the onset of a running program in basic recruit training, it responds by remodeling its cortex, that is, by moving bone mineral from areas of low strain to areas of high strain. Thus, the lesion is not a mechanical failure of the bone, but an area of porosis.[115] The stimulus for the remodeling appears to be more related to the cyclic nature of the stress rather than its intensity.

Foot Blisters

In military populations, foot blisters are a prevalent, although less serious, type of injury associated with exercise.[104,116] It is a common cause for reporting to sick call during basic training and after loaded road-marching in experienced troops[117]; severe cases can cause several days of limited duty in affected individuals. Blisters occur from increased friction between the sock and the skin and are exacerbated by wetness. Improved sock material and use of antiperspirants[118] are being studied.

Cardiovascular Disease

Because physical training and fitness standards are emphasized, and in some cases required, at all levels of military service, there has been increased concern with exertion-related cardiac morbidity and mortality. The concern about undetected risk for heart disease was originally developed from autopsy reports of young soldiers during World War II,[119] and the U.S. Army's decision in the early 1980s to require fitness testing at all ages resulted in The U.S. Army Surgeon General's Office developing an over-40 cardiovascular risk screening program.[120] This program was designed to identify those individuals who were at significant risk, or had evidence of significant cardiovascular disease, before they underwent biannual physical fitness testing. This cardiovascular risk screening program is now integrated into the periodic physical (medical) examination (every 5 years) of all over-40 aged personnel on active duty in the army.[121] The screen is based on the seven risk factors identified in the Framington Heart Study,[122] consisting of age, gender, smoking habit, BP, resting electrocardiogram, glucose tolerance, and total serum cholesterol. A risk index (from the American Heart Association, Coronary Risk Handbook Publication 70-041-A, 1979) of 5% or more (likelihood of developing cardiovascular disease over a 6-year period) requires further medical evaluation before being cleared for physical fitness testing. The over-40 cardiovascular risk screening program was based on research carried out with U.S. Army populations of active duty personnel.[123-127]

The prevalence of risk factors and disease in military populations continues to be an active area of study.[128-131] Sudden death during exercise has various etiologies, with cardiovascular factors as one of the more prominent causes. Drory and coworkers[132] reported that cardiovascular factors accounted for 50% of sudden exertion-related deaths studied in the Israeli Defense Forces between 1974 and 1986. Exercise and sudden death have also been extensively studied in civilian populations.[133]

Exertional Rhabdomyolysis

Acute rhabdomyolysis is a condition that has historically been related to military recruit physical training.[134,135] This injury syndrome is characterized by myoglobinuria, muscle pain, weakness, and soreness. It occurs with the sudden onset of intense or excessive exercise, a situation that has previously been common in military recruit training. It can be prevented by a graduated program of exercise intensity in those recruits who are unfit or inactive. Severe rhabdomyolysis can have fatal consequences if it progresses to renal failure secondary to marked myoglobinuria and tubular necrosis. In 1988, cases of death and hospitalization due to exertional rhabdomyolysis (brought on by inappropriately sudden and intense physical training) were reported[136] in police trainees in New York City and the state of Massachusetts. This suggests that the risk for this condition for military recruit training deserves continued awareness and surveillance.

Environmentally Caused Injuries

Exercise in extreme environments, whether it be physical training or physical job tasks, may lead to injuries. The risks for injury provided by high and low temperatures, high altitude exposure, and atmospheric pollution will be briefly described. For more detailed discussion on these topics, the reader is referred to Pandolf and colleagues[137] and Vogel and colleagues.[138]

High Ambient Temperature

Heat stress is the most common environmental problem with which the exercising individual is confronted. The body is well adapted to dissipate the metabolic heat produced by exercise, but these processes may be severely compromised when they must work against high external (ambient) heat load or humidity loads, or both.

Internally generated heat is brought to the skin surface by increased C.O. and peripheral blood flow and is dissipated from the skin through radiation, convection, and conduction, and by the production and evaporation of sweat. High ambient temperature and humidity reduce the skin-to-air moisture gradient, thereby reducing the effectiveness of convection and sweat evaporation.

The body responds acutely to heat stress by increasing skin blood flow through regional redistribution of C.O. During chronic exposure to heat, a process of acclimatization occurs. This consists of an expansion of plasma volume, increased sweating, and improved circulation.

The risk of heat injury can be reduced by avoiding ambient conditions to which an individual has not acclimatized. Such conditions are best monitored with a wet-bulb globe temperature (WBGT) index:

$$\text{WBGT} = (0.7\ \text{Tnwb}) + (0.2\ \text{Tg}) + (0.1\ \text{Tdb})$$

where Tnwb is the natural wet-bulb thermometer temperature, Tg is the black-globe thermometer temperature, and Tdb is the ordinary dry-bulb thermometer temperature. Risk index zones based on the WBGT and exercising intensity are readily available from preventive medicine officers, physical training publications, or DA Technical Bulletin Med 507.[139]

Heat injuries include the following conditions:

1. Heat cramps: the benign cramping of muscles due to an imbalance of sodium and potassium resulting from salt loss through heavy sweating during exercise in the heat. Heat cramps are treated by fluid and salt replacement.
2. Heat rash: the benign prickling sensation of the skin during prolonged sweating.
3. Dehydration and salt depletion: conditions not exclusively found with heat stress. Dehydration is indicated by decreased urine production, lethargy, anxiety, and irritability. Severe cases result in irregular gait and altered consciousness. Symptoms of salt depletion are similar to those of dehydration in its mild form. Severe forms can result in vomiting, muscle cramps, and eventually, seizures, coma, and death.
4. Heat exhaustion: potentially serious dehydration and salt depletion, but without tissue damage. Heat exhaustion is recognized by the symptoms of headache, dizziness, pallor, nausea, vomiting, uncoordinated gait, and moderately elevated body temperature. It is treated by removing the injured from the hot environment, replenishing salt and fluids, and if necessary, actively cooling the body.
5. Heat stroke: cases of heat exhaustion that have progressed to the point where thermoregulation fails and body temperature rises above 40ºC. Medically supervised aggressive measures of active cooling and repletion of electrolytes and fluid are required.

Low Ambient Temperature

Injury during exercise in the cold is much less a problem than heat induced problems, because clothing can be added to help retain the metabolic heat that is generated. Risk of injury still remains for exposed or wet skin or extremities. Physical training can continue even in severe cold as long as adequate protection to the body surface is provided. Precaution should be taken not to "overheat" during exercise in the cold, as this will produce excessive sweating and wet skin. The latter will lead to rapid and possibly dangerous cooling once exercise ceases.

Cold injury to the inappropriately clothed or accidentally exposed individual can include

1. Hypothermia: a serious condition in which body core temperature falls excessively to impair normal body function. This has been known to occur on cold days at the end of long races, such as a marathon. As the core temperature falls, blood is shunted to the core, leading to shivering and cold extremities. If the core temperature continues to decline, cardiac, pulmonary, and nervous functions will be impaired, and eventual loss of consciousness occurs. Mild hypothermia can be treated with surface rewarming, while severe cases require extensive medical care and measures to rewarm the body core.
2. Frostbite: a condition that can occur in the skin of exercising individuals when tissue water crystallizes, and causes subsequent tissue dehydration and damage. High wind-chill factors, either from the wind or the speed of exercise (eg, cycling) will greatly increase the risk. "Frost nip," or a whitening of an exposed area of skin without actual damage, is undesirable, because it may make the area susceptible to future frostbite. Frostbite can be serious and may

require amputation. Early signs include numbness and white or yellowish color of the affected surface. Treatment consists of rapid rewarming of the affected area in water at 40ºC to 42ºC, preferably under medical supervision.

High Terrestial Altitude

Military troop deployments, although not common, can take place at altitudes between 2,500 and 4,000 m, which places individuals at risk for high-altitude illness or injury. These altitudes also result in well known decrements in exercise capacity and physical performance due to the reduced PO_2 and accompanying disturbances in acid-base balance. Acclimatization to high altitude does occur and, therefore, troops should, if possible, ascend gradually to allow time for the acclimatization process to develop prior to exercising at the intended altitude.

High terrestrial altitude injury and illness include

1. Acute mountain sickness: a condition common in rapid ascent to an altitude above 2,500 m is characterized by severe headaches, nausea, vomiting, decreased appetite, and sleep disturbances. U.S. Army operational doctrine employs gradual ascent or intermediate stops, as well as prophylactic use of acetazolamide. Most people recover in 2 to 4 days as acclimatization occurs. Severe cases can be treated by descent or supplemental O_2. A portable hyperbaric chamber (Ganow Bag) has also been found useful. Severe cases may lead to cerebral edema, which must be rapidly and aggressively treated.
2. High-altitude pulmonary edema: a condition characterized by fluid leaking into the lung as the result of tissue hypoxia. Symptoms include cough, shortness of breath, and cyanosis of peripheral areas. This is a serious condition, which, if not treated with O_2 and descent, can lead to coma and death.

CONCLUSION

Physical fitness is an important goal of military training; it enables soldiers to optimally perform their physically demanding duties. Understanding the physiology of cardiovascular and muscular conditioning is necessary for the development and implementation of optimal training programs for soldiers.

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Chapter 14

PHYSIATRY: INTERDISCIPLINARY MANAGEMENT

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INTRODUCTION

HISTORICAL PERSPECTIVE

THE INTERDISCIPLINARY TEAM
  Kinesiotherapist
  Occupational Therapist
  Orthotist and Prothetist
  Physiatrist
  Physical Therapist
  Psychologist
  Recreation Therapist
  Rehabilitation Nurse
  Speech and Language Therapist
  Social Worker
  Vocational Rehabilitation Counselor
  Patient and Family

THE INTERDISCIPLINARY TEAM AT WORK
  Case Study 1: Distal Femur Fracture
  Case Study 2: Above-Knee Amputation
  Case Study 3: Traumatic Brain Injury
  Case Study 4: Spinal Cord Injury
  Case Study 5: Cervical Strain

CONCLUSION

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INTRODUCTION

Advances in medical technology and in emergency health service healthcare delivery have brought about a multidisciplinary approach to patient treatment throughout medicine. The physician, whether in a hospital emergency department, his own office, or a battalion aid station, is surrounded by other healthcare practitioners and technicians who possess skills and knowledge that are essential for the optimum care of the patient. Even though multidisciplinary teams are common, a true interdisciplinary approach to healthcare delivery remains rare. An interdisciplinary approach requires the communication of observations and impressions, followed by the preparation of comprehensive goals and a treatment plan. The underlying difference between the multidisciplinary approach and the interdisciplinary approach is communication.

The common multidisciplinary approach may involve many practitioners who set independent treatment goals and make their own treatment plans. In the interdisciplinary approach, each practitioner does his own assessment and reports his findings, impressions, and recommendations to other professionals during the "team conference." Through the mechanism of the team conference, information is shared, common goals are established, and a consolidated treatment plan is formulated. This communication ideally leads to an outcome which will be greater than the sum of its individual parts.[1]

Although the emphasis of this chapter is on an interdisciplinary approach within the military healthcare system, permanently impaired soldiers may receive some or all of their rehabilitative care within the Veterans Affairs healthcare system or in civilian rehabilitation facilities.

HISTORICAL PERSPECTIVE

The response of military medicine to war injuries has played a key role in the development of rehabilitation medicine as a specialty and in the development of the individual disciplines whose practitioners make up the interdisciplinary rehabilitation team. Between 1890 and the onset of World War I, an area of specialization within medicine developed which was known as electrotherapy. Electrotherapists frequently aligned themselves with homeopathic physicians, hydrotherapists, and others who espoused the use of natural treatments, rather than the often detrimental purgatives and mercurials used by the allopathic physicians of the day. With time the venue of the electrotherapists (or electrotherapeutists, as they became known) expanded to include exercise, dietetics, psychotherapy, climatology, and phototherapy. The interest of electrotherapeutists in the use of physical modalities resulted in these physicians being referred to as "physical therapists" or "physiotherapeutists."[2]

At the same time, a subspecialty of surgery called orthopedics was evolving. The orthopedists distinguished themselves by the use of mechanical devices, such as braces, in the treatment of patients. In 1917, with America's entry into World War I, it became apparent that the U.S. Government would have to care for large numbers of battlefield casualties, and a war preparedness committee was organized. Joel Goldthwait, an orthopedist and director of the mechanical department at the Massachusetts General Hospital, urged his personal friend, General Pershing, to create a division of orthopedic surgery in the army. In August 1917, the new Division of Special Hospitals and Physical Reconstruction was created. This division included general surgery, orthopedic surgery, head surgery, and neuropsychiatry. Orthopedics was to be responsible for the construction of artificial limbs. Mary McMillan, who was then chief aide to Dr. Goldthwait, was given the responsibility for training "orthopedic aides." Under her direction, 800 women underwent 30-day training courses in "military massage." Later, this group would become the nucleus of physical therapy technicians, from which evolved the American Association of Physical Therapists.[2]

Also during 1917, Frank Granger was appointed head of the physiotherapy section of the Division of Special Hospitals. Dr. Granger was president of the American Association of Electrotherapy and Radiology at that time; thus, physiotherapeutic physicians became recognized in the area of physical reconstruction.

The discipline of occupational therapy originated (before either the physician physiotherapeutists or Mary McMillan's physical therapy technicians) as the "work cure and moral treatment" therapists of 19th century psychiatry. In 1918, the discipline of occupational therapy was added to the staff of the Division of Special Hospital and Physical Reconstruction, completing the nucleus of the rehabilitation team as we know it today.[2]

In the years between World Wars I and II, the physiotherapeutists continued to promote the use of physical modalities and were instrumental in developing training programs and accreditation measures for both the occupational and physical therapists.[2]

Because the onset of World War II occurred at a time of advancing medical technology, greater numbers of war injured were saved than was previously possible. Dr. Howard Rusk was appointed head of the U.S. Air Force Convalescent Training Program, and is credited with developing modern rehabilitation medicine and, after the war, with founding its first institute. By 1946, the term "physiatrist" was coined to designate a physician specializing in physical medicine. In 1947, the Advisory Board for Medical Specialties and the American Medical Association established the American Board of Physical Medicine to be responsible for conferring certification in physiatry, and in 1949, the words "and Rehabilitation" were added to the Board's title.[2]

Examination of the historical development of physical medicine and rehabilitation makes it easy to see the roots of the interdisciplinary team. In a way, the core disciplines of physiatry, physical therapy, and occupational therapy "grew up together." It is unlikely that any of them could have developed alone. Other disciplines that are important members of the team, but whose history has not been specifically discussed, were evolving alongside the early electrotherapeutists and orthopedists. They will be addressed in the next section.

THE INTERDISCIPLINARY TEAM

The composition of a rehabilitation team varies depending on the characteristics of the patient's injuries and resulting impairments. The ultimate goal of the team is always the best possible functional outcome in the least possible time. In the case of simple injuries, such as an uncomplicated fracture, the team may consist of a physician and a physical therapist. The physician is responsible for making the diagnosis (a fracture) and applying a medical treatment (a cast). A treatment plan is formulated by the physician and therapist, and the therapist is responsible for training the patient in the treatment plan and its restrictions. The therapist is also responsible for identifying and remediating any concurrent disabilities (eg, decreased mobility, which requires training in crutch ambulation). More complex injuries or permanent impairment requires more team members.

Kinesiotherapist

Kinesiotherapy, previously called corrective therapy, is a discipline unique to the Veterans Health Service. During World War II, the shortage of physical therapists led to the recruitment of physical educators to provide exercise therapy for psychiatric patients. These individuals became known as corrective therapists. Recently, the discipline has adopted the title kinesiotherapist to emphasize its interest in functional movement.[3] Kinesiotherapists provide exercise programs to improve and maintain gross motor function; evaluate and train patients for adapted automobile driving; and promote fitness through exercise for acute, chronic, and convalescent patients.[1]

Occupational Therapist

As already described, occupational therapists were added to the corps of rehabilitation aides in 1918. Many presidents of the Occupational Therapy Association were physical therapy physicians, and training for occupational therapists progressed under their leadership. In 1932, the training was increased from 6 months to 9 months, and at present there is a general prerequisite for a baccalaureate degree and a certificate of occupational therapy training.[2]

The role of the occupational therapist on the rehabilitation team includes evaluation and training of the patient in activities of daily living (ADL), such as feeding, bathing, dressing, bathroom transfers and activities, homemaking, and child care skills. Occupational therapists evaluate and supervise activities aimed at improvement and maintenance of joint range-of-motion, muscle strength, endurance, and coordination of the upper extremities as relates to functional tasks. Occupational therapists also identify and remediate sensory and cognitive deficits. They evaluate the patient's home environment and recommend modifications and equipment that will maximize independent functioning. The occupational therapist educates and trains the patient and his family in adaptive techniques and potential for independence and helps the patient explore vocational skills and avocational interests. Often, occupational therapists are responsible for the fabrication of thermoplastic and plaster splints that are used to support and protect paretic limbs and prevent deformities. In some settings, occupational therapists are involved in the evaluation of driving habits and skills and the evaluation and management of dysphagia. Overall, the emphasis of the occupational therapist is on the development of functional skills and abilities.[1]

Orthotist and Prosthetist

An orthotist is trained in the design and construction of a wide variety of braces (orthoses). A prosthetist is trained in the design and construction of artificial limbs (prostheses). To sit for the certificate examination in either of these professions, the candidate must have a baccalaureate degree and 1 year of apprenticeship. Professionals from both disciplines work as part of the rehabilitation team to provide the appropriate devices for maximizing patient function and minimizing secondary disability.

Physiatrist

A physiatrist is a physician who has completed specialty training in Physical Medicine and Rehabilitation. Currently, a Doctor of Medicine or Doctor of Osteopathy degree followed by 1 year of general internship training, 3 years of specialty training, and 1 year of independent practice is required to sit for the American Board of Physical Medicine and Rehabilitation.[4] In most rehabilitation settings, the physiatrist acts as the team leader. By virtue of his training, the physiatrist has a broad understanding of the expertise and roles of the other team members, as well as his own medical understanding of disease processes and effects. He has the ability to take all the team's information and synthesize a comprehensive clinical picture and plan. The physiatrist can also act as liaison to other physicians involved in the patient's care, but who may not understand the function of the interdisciplinary team.

Physical Therapist

Physical therapy training, like occupational therapy training, has progressed from McMillan's 30-day course for physical therapy technicians, to programs requiring a baccalaureate degree, a certificate in physical therapy, and a state licensing examination.[2] Physical therapists provide patient training and supervision in all forms of exercise programs (range-of-motion, resistive, endurance or aerobic, coordination) and mobility techniques (transfer from one position to another, ambulation with or without assistive devices, wheelchair mobility). Physical therapists are responsible for the administration of physical therapy modalities (heat, cold, hydrotherapy, massage, traction, electrical stimulation) and the assessment of adaptive equipment needs (crutches, canes, walkers, wheelchairs, and wheelchair cushions).[1]

Psychologist

The role of the psychologist is, perhaps, one of the most diverse on the rehabilitation team. In the case of a patient who has impairment of cognitive processes, the psychologist or neuropsychologist administers tests of intelligence, memory, and perceptual functioning to define the deficits and help the team plan treatment that will compensate for these deficits. Often, patients who appear to be functioning well have subtle cognitive deficits that will interfere with their participation in the rehabilitation program. The patient without cognitive deficits may benefit from psychological counseling regarding his adjustment to changes in his physical body and to his role within his family and community. The psychologist can be helpful to the patient's family in these same areas. Just as important, the psychologist may also act as counselor to the rehabilitation team by identifying nonproductive interactions between the team and the patient, between the team and the family, or between team members.

Recreational Therapist

Leisure functioning, or recreation, is easily recognized as a component of a rewarding lifestyle, and the need for recreation is not blunted or eliminated by physical impairment or disability. As with the other disciplines within the rehabilitation team, recreational therapy has evolved over the years and, at present, is best defined as the use of recreational services to bring about desired changes in behavior and promote the growth and development of the patient. Currently, undergraduate- and graduate-level training programs are accredited, and a voluntary certification examination is recognized by several state and local agencies as well as other private institutions and organizations.[5]

Rehabilitation Nurse

The rehabilitation nurse performs all usual nursing duties, but has the additional responsibility of promoting maximal function and independence of the patient. While rehabilitation nursing does not necessitate the acquisition of new knowledge, skills, or attitudes, a greater depth of understanding is required in some areas. Specifically, the rehabilitation nurse must have a full understanding of the psychosocial effects of long-term illness; a strong knowledge of anatomy, physiology, and pathophysiology of the neuromuscular system; and an understanding of kinesiology (the science of body movement). In addition, the attitude and temperament of the rehabilitation nurse must differ in some ways from that of the acute care nurse. The rehabilitation nurse must deal with the chronicity of physical impairments, and with a role shift from caring and doing for the patient to assisting the patient to care for himself.[6] The rehabilitation nurse works with the patient outside the formality of the therapy departments and during the evening and early morning hours, and is in the best position to assess the patient's learning and the carry over of new skills and abilities.

Speech and Language Therapist

The responsibilities of the speech and language therapist (or speech therapist) include all aspects of communication. A speech therapist evaluates the patient's cognitive and language skills (the ability to comprehend as well as the ability to produce speech), including written and other nonverbal modes of communication. The speech therapist is an expert in the mechanical function of the oropharyngeal and laryngeal areas and will assess the patient's ability to chew and swallow, as well as to perform the mechanical activities involved in speech. When verbal communication is not possible, the speech therapist will educate the patient, family, and other caregivers in alternative forms of communication such as sign language or the use of adaptive devices (word and letter boards, electrolarynxes, speech synthesizers). Since communication and cognition are closely linked, the speech therapist is also involved in cognitive evaluation and retraining.

Social Worker

The social worker interacts with the patient, family, and team to gather information regarding the patient's living situation and resources, including financial status. The social worker has a working knowledge of programs and community support services that may be useful to the patient. In some institutions, the social worker also assumes the responsibility for patient and family counseling.

Vocational Rehabilitation Counselor

Vocational rehabilitation counseling developed in conjunction with rehabilitation medicine at the time of World War I. Over the years, educational requirements for this discipline have evolved to include civil service certification and a master's level degree in psychology, education, personnel administration, or rehabilitation counseling.[7]

The responsibility of the vocational rehabilitation counselor is to work with a physically handicapped or mentally impaired patient to establish a mutual understanding of capabilities and limitations through review of records, testing, and direct observation. With this information, the vocational rehabilitation counselor assists the patient in developing appropriate vocational plans and goals and in obtaining the necessary training to achieve these goals. The vocational rehabilitation counselor also acts as an advocate for the patient in the workplace.[7]

Patient and Family

The team, no matter how expert or dedicated, can merely direct and instruct the patient. Only the patient can perform the exercises, learn the techniques, and make the decision to adapt to his impairment. The team can advise and instruct the family regarding the patient's disabilities and capabilities, but, ultimately, it is the patient and his family who determine the success of the team's efforts.

THE INTERDISCIPLINARY TEAM AT WORK

To illustrate the function of an interdisciplinary rehabilitation team, several case studies, ranging from a simple long-bone fracture to the more complex case of traumatic brain injury are presented here.

Case Study 1: Distal Femur Fracture

A 22-year-old soldier sustained a distal femur fracture in a motor vehicle accident. He underwent immediate reduction and intramedullary rod placement, and the extremity was placed in a long-leg cast with the knee in full extension. The patient was referred for rehabilitation medicine evaluation and management on the first postoperative day. The orthopedic surgeon indicated that the patient was to be non–weight-bearing on the fractured right leg for 6 weeks.

The physiatrist's evaluation revealed that in addition to the current injury, the patient had an injury to the left hand that had occurred while playing high school football. The left hand still caused the patient discomfort, especially when doing pushups. On physical examination it was noted that the patient had tenderness to palpation over the scaphoid bone of the left hand. The physiatrist ordered a radiograph of the left hand and wrist.

The patient was referred to physical therapy for strengthening of the bilateral upper extremities; left lower extremity; and right hip flexor, including extensor and abductor progressive resistive exercises and isometric exercise of the right quadriceps, hamstring and ankle plantar and dorsiflexor muscle groups. The physical therapist began training the patient in bed mobility and transfers while avoiding weight bearing on the right lower extremity. Training in crutch ambulation was delayed pending the left hand radiographs. The patient went to occupational therapy for assessment of self-care skills (bathing, dressing, and bathroom activities) while maintaining non–weight-bearing on the right lower extremity.

The social worker evaluated the patient's social support systems, discharge plans, and functional requirements for discharge, such as whether he would need to ascend and descend stairs in his home, or if he would need to cook for himself.

The initial rehabilitation team conference was held 4 days after the patient's referral to the Rehabilitation Medicine Service. The physical therapist reported that the patient had mastered his exercise program quickly and was able to execute the program independently. The patient was also independent in transfer skills, but had complained of pain in his left hand when attempting to bear weight on his left palm during transfers. This problem had been alleviated by the patient's bearing weight on his knuckles instead of his palm during transfers. The occupational therapist had provided the patient with a dressing stick, a long-handled shoehorn and an elastic shoelace for his right shoe, and he was now independent in dressing activities. He was also independent in bathroom activities and transfers, and had prepared a cold meal in the kitchen. He had been discharged from occupational therapy.

The physiatrist reported that the radiographs of the left hand had shown a nonunion of a scaphoid bone fracture. The Orthopedic Hand Surgery Services had been consulted about the hand injury and reported that no intervention was currently indicated. They also said that while weight-bearing on the hand would not be detrimental, it would be uncomfortable. This limitation would interfere with standard crutch ambulation. The social worker reported that the patient could return to his parents' home for convalescence, but that would necessitate his climbing a flight of stairs with a railing on the left-hand side (ascending) to reach the bathroom and his bedroom. Since his mother was a full-time homemaker, he would not be required to perform any homemaking tasks. The goal of independent ambulation, with the appropriate assistive devices (including stairs), was set in compliance with his weight-bearing limitations. This was to be accomplished by serial trials of axillary crutches or, if this was not tolerated, a Loftstrand crutch with a forearm support on the left and a standard Loftstrand crutch on the right.

At the next team conference, 1week later, the physical therapist reported that ambulation with axillary crutches had not been tolerated due to left-hand pain. Attempts at ambulation with a left forearm support crutch and a right Loftstrand crutch had been inadequate due to the patient's inability to swing his right leg through without putting weight on it. After consulting with the physiatrist, a 0.5-in. lift was placed on the left shoe by the orthotist. With the crutches and his shoe lift, the patient was then able to ambulate unlimited distances and ascend and descend stairs safely. The patient was discharged to his home for 5 weeks of convalescent leave.

Upon return from convalescent leave, a radiograph of the right femur showed satisfactory healing, and the cast was removed. The orthopedic surgeon cleared the patient for range-of-motion of the right knee, and weight bearing as tolerated on the right lower extremity. Initial physiatry evaluation noted active and passive right knee motion from full extension to 20° of flexion. There was approximately 1.5 inch of right calf and thigh atrophy compared to the left side. Right ankle range-of-motion showed normal plantar flexion but only 5° of dorsiflexion. Muscle strength of the ankle dorsiflexors, knee extensors and knee flexors was graded as 4/5.

The patient was referred to physical therapy for (*a*) active assisted range-of-motion of the right ankle and knee using moist heat and/or ultrasound to facilitate soft tissue extensibility; (*b*) progressive resistive exercise of the right quadriceps, hamstring, and ankle dorsiflexor muscle groups; and (*c*) progressive ambulation with weight bearing as tolerated. The social worker was consulted to act as liaison to the patient's military unit and to determine whether return to limited duty or further convalescent leave would be appropriate.

At the team conference 5 days after readmission, physical therapy reported that the patient had normal right ankle range-of-motion and ankle dorsiflexor strength. His knee range-of-motion had progressed to 80° of active flexion. Quadriceps strength remained decreased, but was improving. The shoe lift had been removed from the left shoe and the patient was ambulating for unlimited distances with his forearm support crutch on the left. The social worker reported that the patient's unit did not have an appropriate light-duty position for him, but that the patient would be temporarily reassigned and would be doing clerical work that required minimal ambulation. He could thereby live in the hospital barracks and attend physical therapy as an outpatient on a daily basis. The patient was discharged to the barracks and the order given for continued physical therapy.

The patient attended physical therapy daily as an outpatient and, under the direction and advice of his physical therapist, augmented this activity with resistive exercises in the base gymnasium and swimming pool. The physical therapist consulted with the physiatrist about the advisability of allowing the patient to resume a running program. After discussion with the orthopedic surgeon, the patient was cleared to resume running as tolerated. At the Rehabilitation Medicine Clinic followup 4 weeks after discharge, isokinetic testing showed the patient had full right knee range-of-motion, and normal and symmetric hamstring and quadriceps strength. He was ambulating without assistive devices and had begun running under the direction of his physical therapist. He was given a 3-month temporary physical profile restricting his running and marching activities initially, then allowing a gradual increase in these activities. He was returned to his unit.

The patient was seen for followup 3 months later at the time his profile was to expire. He reported he was able to run 5 miles at a pace sufficient to pass his physical training test, and could walk or march unlimited distances. He continued to have some discomfort in his left hand when doing pushups, but not to a significant degree. The patient was returned to duty without restrictions and discharged from the Rehabilitation Medicine Service.

Comment

In this example, multidisciplinary management (where healthcare professionals from different disciplines set discipline-specific goals without forming a comprehensive, coordinated plan) could have resulted in subtle, yet important, impairments being overlooked. For instance, if the nonunion of the scaphoid fracture had not been diagnosed and accommodated, the patient's mobility might have been severely limited, resulting in secondary deconditioning, which would have required more rehabilitation later. He also might have become a disposition problem if he could not ambulate with crutches and climb stairs. In addition, the therapists might have interpreted the patient's difficulty with crutch ambulation as malingering, thereby destroying the therapeutic relationship and further impeding recovery.

The communication between team members allowed them to consider the possibility of multiple secondary complications. Atrophy of the right leg musculature was limited by the early initiation of isometric exercises. Later, the potential complication of limited knee range-of-motion was avoided by close supervision of the rehabilitation process once the cast was removed. Finally, while he was preparing for return to full duty, which included physical training, the patient was protected from accusations of malingering by his physical profile. Without communication between the team members, this soldier could have become a medically discharged, permanently impaired, angry veteran.

Case Study 2: Above-Knee Amputation

A 19-year-old reservist was activated during the Persian Gulf War and sustained multiple fragment injuries during a missile attack. The most severe injury was to the left proximal leg, but other fragments involved the right distal thigh and sciatic nerve, the right distal arm and ulnar nerve, and the left brachial plexus. There was also a fragment in the area of the left ischial tuberosity. The patient was evacuated to a local hospital where the wounds were debrided. Vascular supply to the left foot and leg was severely compromised, but repair was attempted anyway. The patient was transferred to an army medical center for definitive care.

Evaluation at the medical center revealed that, due to inadequate vascular supply and extensive soft tissue loss, the left lower extremity was not salvageable. The wounds over the left ischial tuberosity, the right elbow, and right thigh were open, but healing by secondary intention. Neuromuscular examination was consistent with a distal lesion of the right sciatic nerve, a lesion of the right ulnar nerve at the elbow, and weakness of upper brachial plexus innervated muscles on the left. Rehabilitation Medicine Service was consulted for recommendations regarding the level of left lower extremity amputation, evaluation of peripheral nerve injuries by electrodiagnostic testing, and comprehensive rehabilitation management.

The physiatrist advised that preservation of maximal stump length should be the goal of amputation. Although a knee disarticulation stump presented certain problems with prosthetic fit and cosmesis, the extra length of the stump would provide some mechanical advantages over a shorter, above-knee amputation. Electrodiagnostic testing of the right lower and both upper extremities was interpreted as showing a partial right ulnar nerve lesion just above the elbow, a partial right sciatic nerve lesion in the distal thigh, and a probable neuropraxic lesion of the left upper brachial plexus. Left below-knee amputation was attempted, but the limb distal to the knee did not appear to be viable at the time of surgery, so a knee disarticulation was performed.

Postoperatively, the patient was transferred to the Rehabilitation Medicine Service and was ordered to occupational therapy for

- bilateral hand evaluations to include manual muscle testing along with sensory and dexterity evaluation;
- splinting of the right hand to prevent claw deformity and flattening of the palmar arch;
- facilitative techniques, such as electrical stimulation, to enhance recovery of right hand and left shoulder girdle musculature; and
- assessment of and training in ADL.

The patient was referred to physical therapy for

- evaluation and training in bed mobility and transfers;

- wheelchair adaptation to allow independent mobility;
- isometric and isotonic exercise of the hip and thigh musculature bilaterally;
- splinting of the right ankle to prevent plantar flexion contracture;
- active assisted range-of-motion and resistive exercises to all four extremities as tolerated; and
- facilitative techniques, such as electrical stimulation, to encourage activation of the distal right lower extremity muscles.

The physical therapist also taught the patient about amputation stump wrapping to reduce edema and sensitivity, and prone lying to prevent hip flexion contracture. The psychologist evaluated the patient and his family's response and adjustment to the injury, while the social worker evaluated the patient's support systems and his social and work history, and acted as liaison between the patient, his family, and the military administrative system.

At the initial team meeting 10 days following amputation, the physiatrist reported that the patient was medically stable and his amputation and other wounds were healing well. The occupational therapist reported that the patient had moderate deficits of strength and sensation in the ulnar distribution on the right, which required use of adaptive devices for writing, but which otherwise were not interfering with functional activities. The left shoulder girdle strength was improving. The patient was independent in self-care activities and transfers. It was noted that, while he cooperated and carried out all instructions and requests, the patient appeared to be withdrawn and angry, and resisted attempts of the therapist to involve him in conversation. The physical therapist reported that left hip range-of-motion was normal and hip extension and abduction strengths, while limited by postoperative pain, appeared to be improving. The patient still required assistance with stump wrapping and was reluctant to look at or touch his stump. He required frequent reminders to lie prone. Left ankle range-of-motion had normalized, and he had developed 2/5 ankle dorsiflexion and eversion strength, and 4/5 plantar flexion strength. The patient was independent in transfers and had been provided with a wheelchair with a right-hand rim with oblique projections to compensate for his hand weakness. He had been given a Roho cushion to minimize the pressure over the ischial wound on the left. He could propel the wheelchair independently for unlimited distances. The physical therapist also noted that the patient appeared withdrawn. The rehabilitation nursing staff reported that the patient was capable of being independent in self-care activities, but frequently requested that things be done for him. The nurses noted that the patient's family fostered his dependence and were demanding and critical of the nursing staff.

The social worker reported that the patient had been employed as a manual laborer prior to his injury and had joined the reserves to supplement his income. He had been living with his parents in a single-story home and was engaged to be married. Due to his military activation and the subsequent injury, the wedding had been postponed. Furthermore, the patient had an older brother who was a Vietnam veteran and a recovered substance abuser. The brother was receiving disability benefits for posttraumatic stress disorder. This same brother was active in veterans' groups and expressed a great deal of anger toward the military and veterans' healthcare systems because the diagnosis and treatment of his stress disorder had been delayed for several years. The psychologist confirmed that the patient was angry about disruption of his own life by his military duties, and that he had never truly considered the possibility of activation when he joined the reserves. He was reacting to his own anger and his brother's anger by dependence and withdrawal from socialization. He was not, however, exhibiting any vegetative signs or symptoms of depression.

On the basis of the information presented by the team members, goals were set for the next 10-day period, including ambulation with assistive devices and contact guard assistance, compliance with exercise programs, improved independence on the ward, completion of prevocational evaluation, and improvement in affect and socialization. Physical and occupational therapy programs were continued by beginning ambulation using a walker with a right forearm support and with a right molded-plastic ankle-foot orthosis. The psychologist worked with the patient individually and enrolled him in a support group for Persian Gulf War casualties. The physiatrist began collecting the necessary information for completion of the patient's medical evaluation board (MEB) and kept both the patient and his family informed on the status of these proceedings. The social worker continued to be liaison for the patient and his family, and attempted to make the family aware of some of the detrimental effects of the brother's anger and negativity, and of their fostering the patient's dependency. The nursing team, while understanding the patient's difficulty with adjustment, continued to encourage independence. The occupational therapist began prevocational assessment to define the patient's areas of interest and skills. The patient was referred to recreational therapy to explore avocational interests and improve socialization.

Ten days later, at the next team meeting, the rehabilitation nurse reported that the patient still tended to be withdrawn but was now independent in all self-care activities. The physical therapist reported that the patient's right ankle dorsi and plantar flexor strength now showed only minimal deficits, and that he was ambulating independently with axillary crutches but used the wheelchair for long distances. The surgical staples had been removed from the stump and it was well healed, conical, and nontender. He continued to have some phantom sensation, but no phantom pain. The occupational therapist reported that he had persistent, intrinsic muscle weakness in his right hand, but the left shoulder girdle strength was now normal. Prevocational assessment had revealed an interest in, and aptitude for, work with computers and information management. The psychologist reported that the patient was participating in individual and group therapy and, although there was still a good deal of residual anger, he was beginning to talk about and plan for his future in a realistic fashion. The social worker reported that his MEB had been completed and the physical evaluation board (PEB) was in process. His brother remained hostile but had agreed to refrain from criticizing his brother's care in front of the patient. The physiatrist reported that all the fragment wounds had healed, but the scar over the ischial tuberosity would be problematic for fitting with a standard quadrilateral socket above-knee prosthesis. At this point, it was decided to fit the patient with a temporary plastic quadrilateral socket with Silesian band suspension, safety knee, and single-axis foot.

Within 3 weeks the prosthesis was fabricated and the patient achieved independent ambulation using a single point cane. With ambulation the patient's stump began to shrink and daily adjustment of stump socks was required. Administratively, the patient had been discharged from the military with 50% service connected disability. It was felt by the physiatrist and prosthetist that the patient was not yet ready for a definitive prosthesis, but was ready for discharge from the hospital. The patient and his family wished to return home, which was several hundred miles from the military medical center. The physiatrist contacted the Rehabilitation Medicine Service at a Veterans Affairs Medical Center (VAMC) near the patient's home and transferred the patient's records and care to the VAMC.

The patient was seen by the VAMC Rehabilitation Medicine Service 1 week after discharge from the army medical center. An area of skin breakdown was noted over the ischial tuberosity on the right, and the patient continued to have right hand intrinsic muscle weakness in the distribution of the ulnar nerve. The ischial sore was treated with hydrogen peroxide rinses, rest periods with the prosthesis off during the day, and Duoderm dressing when wearing the prosthesis. The patient was continuing to add stump socks, currently wearing 15-ply, and was independent in ambulation without assistive devices. He was referred to the VAMC hand surgery clinic for follow-up of his ulnar nerve injury, and to the social work service for continued patient and family support. The vocational rehabilitation counselor continued testing and assistance with vocational training.

The patient was seen in VAMC Rehabilitation Medicine Services clinic at 2-week intervals and was discussed in outpatient team meetings. The social worker reported that the patient was proceeding with plans for his wedding. He was still reluctant to talk about his injuries, but he appeared to be adapting to his return to civilian life. The vocational counselor reported that he had narrowed his career choice to computer programming and was exploring training options. The physiatrist noted that the hand surgery clinic had requested repeat electrodiagnostic evaluation of the right ulnar nerve and that this testing had shown some improvement in nerve function, but had also shown significant residual axonal loss and denervation. The size of the knee disarticulation stump had stabilized and the patient was ready for permanent prosthetic fitting.

At 6 weeks following discharge from the army medical center, the patient was seen in conjunction with the prosthetist at the VAMC Rehabilitation Medicine Service clinic. It was noted that the scarred area over the right ischial tuberosity continued to be poorly pressure tolerant. It was decided to fit the patient with a narrow, medial lateral socket, which would eliminate weight bearing on the ischial tuberosity. The prosthesis was fabricated, and the patient was admitted to the VAMC rehabilitation unit for prosthetic adjustment and gait training. He quickly adjusted to the new prosthesis and was discharged after 10 days. He was followed in the VAMC rehabilitation medicine clinic at increasing intervals and continues to be seen annually, or on an as-needed basis for prosthetic evaluation.

Comment

In this case, early interdisciplinary intervention allowed for optimal surgical and rehabilitation planning. Not only was the rehabilitation team allowed to have input into the level of the amputation, but the patient also had the opportunity to be acquainted with the team from the very beginning of his hospital stay.

Once the surgery was over, careful attention to the patient's affect and social situation played a large part in determining an optimal outcome. Without recognition of confounding and conflicting family dynamics, the relationship between the nursing staff and the patient may have become nonproductive or even adversarial. Without close communication between the military rehabilitation physicians and the VAMC physicians, the patient may have been "lost to followup," or the confidence and trust established at the military hospital could have been destroyed by delay and redundancy.

Case Study 3: Traumatic Brain Injury

A 20-year-old soldier sustained facial fractures and closed head injury when his jeep overturned. At the scene he was pulseless and breathless. He was resuscitated and transported to the base hospital before air-evacuation to a civilian, Level I trauma center. On admission to the trauma center, the patient had a Glasgow Coma Scale rating of 4. A computed tomography (CT) scan of his head showed petechial white matter hemorrhages and diffuse swelling. The patient was taken to the operating room where an extraventricular drain was placed.

Postoperatively, the patient required hyperventilation, steroids, and draining of cerebral spinal fluid to control intracranial pressure. He was started on phenobarbital for seizure prevention, and required captopril for management of malignant hypertension. He remained in a coma for 20 days, during which time tracheostomy and gastrostomy feeding tubes were inserted, and he was weaned from the ventilator. Surgical repair of his facial fractures was attempted, but at induction of anesthesia, the patient had an episode of ventricular fibrillation, which necessitated an electrical defibrillation and lidocaine drip.

A myocardial infarction was diagnosed by cardiac enzyme and electrocardiographic changes. Approximately 3 weeks after the injury, the patient began spontaneously opening his eyes and responding to auditory and tactile stimuli. He was medically stable and arrangements were made for his transfer to a VAMC rehabilitation unit near his family's home.

On arrival at the VAMC, the patient was evaluated by a physiatrist who found that he was awake and alert, but did not verbalize or follow commands. He was mildly combative. He appeared to have upper and lower extremity weakness on the left side. He was diffusely hyperreflexic and had bilateral Babinski signs. Cranial nerves could not be evaluated due to the facial fractures and limited patient cooperation. Bilateral ankle dorsiflexion range-of-motion was mildly decreased. There were also 5° knee flexion contractures bilaterally, and a mild decrease in left shoulder range-of-motion. He had tracheostomy and gastrostomy tubes in place. His medications included phenobarbital, captopril, and haloperidol.

The plastic surgery department was consulted for management of the facial fractures; internal medicine for management of hypertension, arrhythmias, and postmyocardial infarction management; ear, nose, and throat surgery for tracheostomy management and possible decannulation; and neurosurgery for followup of head injury and seizure risk. The patient was referred to speech and language therapy for evaluation of swallowing, language perception, and potential for communication by nonverbal means. Psychology Services was consulted for cognitive evaluation and remediation. A social worker was consulted to determine the patient's planned disposition and to act as liaison between the military administration, the VAMC, and the family. The physical therapy department was consulted for active assisted range-of-motion to all extremities, resistive exercises as tolerated, bed mobility, sitting balance, transfers, and progressive ambulation as tolerated. The occupational therapist was consulted for upper extremity strength, sensation and dexterity evaluation, evaluation and training in ADL, cognitive and perceptual evaluation, and remediation. Orders were written for the nursing staff to begin bowel and bladder patterning programs to establish continence. The dietitian was consulted to evaluate nutritional status and recommend nutritional management.

At the initial team conference held 1 week after the patient's admission, the physiatrist reported that a plastic surgery evaluation of the facial fractures was underway and that a CT scan of the face had been scheduled. The nurse interjected that the patient would require sedation for this study. The physician from internal medicine recommended tapering off the captopril since the patient was normotensive, and hypertension had most likely been related to his acute head injury. There was no restriction placed on his activity level. Ear, nose, and throat surgery service had changed the tracheostomy tube to an uncuffed tube but did not recommend decannulation at this time due to continued large amounts of pulmonary secretions and the planned facial surgery. The neurosurgeon had recommended continued seizure prophylaxis for the next 11 months due to the presence of multiple hemorrhages.

The rehabilitation nursing staff reported that the patient did not follow commands, was combative, and required restraint to keep him from removing his feeding tube and tracheostomy. The haloperidol he received as needed for agitation was only partially effective. He was tolerating his tube feedings well. Bowel patterning had been successful, using every-other-day suppositories and digital stimulation, but bladder patterning was only partially successful; 50% of the time he voided when placed on the toilet; and he was incontinent approximately eight times a day. Postvoid residual volumes had been less than 50 cc^3. The dietitian reported that the patient was mildly anemic, protein deficient, and was 15 lb below his ideal weight. He was receiving both calorie and protein supplementation through his gastrostomy tube. The speech and language therapist reported that the patient had been uncooperative with both swallowing and language evaluation. At this time, barium swallow or oral feeding was not recommended. The physical therapist reported that the patient was combative and did not follow commands or demonstration. He neglected his left side. He had independent rolling in bed to the right, but not the left. He had poor sitting balance, falling to the left or backward. He tolerated range-of-motion poorly, but joint range-of-motion was being maintained. He had been fitted with footdrop splints.

The occupational therapist also reported that the patient was combative and did not follow commands. He did some functional activities with his right upper extremity and had some left upper extremity movement in synergy patterns. Cognitive and perceptual evaluation could not be performed due to limited cooperation. He was dependent for all ADL. The social worker reported that the patient was an adopted child who had exhibited behavior problems as an adolescent. Both parents were employed full time and would not be able to care for him at home unless he was completely independent. The psychologist reported that the patient could not be evaluated due to low cognitive level.

After discussion, it was decided that medications might be blunting the patient's cognitive abilities. New treatment would include administering carbamazepine for seizure prophylaxis, and tapering off the phenobarbital. The haloperidol would be discontinued, and the patient would be started on amitriptyline in the evening with hydroxyzine hydrochloride (Vistaril) as needed for agitation. He would not be treated in the open physical or occupational therapy gymnasiums but in private treatment rooms where there would be minimal distraction. In an attempt to reduce the use of medication and restraints, the nursing service would provide one-on-one supervision when the patient was on the ward. Goals of increased attention span and mimicking of activities, increased awareness of the left side, fair sitting balance, 75% urinary continence, and decreased combativeness were set for the next 2 weeks. The physiatrist would order a urine culture to assess possible urinary tract infection that might be contributing to urinary incontinence.

At the next staffing conference 2 weeks later, some improvement in behavior had been manifested as less need for medications and restraint, but no progress had been made in the areas of continence, functional abilities, or cognitive function. The 3-dimensional CT scan had been obtained, and the plastic surgery team was planning a multistage reconstruction. However, the anesthesiologist would not clear the patient for elective surgery until he was at least 6 months postmyocardial infarction. The patient's family was angry and critical of his care and had not yet reconciled to the possibility that the patient might not recover completely. They were anxious for the facial surgery to be done so that the patient would "look normal." The team agreed to maintain the same goals and to review the patient's progress in 2 weeks.

No change or progress was reported at the next team conference. It was decided that the patient was not yet ready to participate in a comprehensive rehabilitation program. The social worker arranged for his placement in the skilled care facility of the VAMC, where he would continue to receive daily occupational and kinesitherapy, and be treated by a speech therapist 3 times a week. He was reviewed in the rehabilitation clinic at 2-week intervals, or whenever there appeared to be a change in his status. A family conference with the treatment team was held to explain the plan and answer any questions.

At the family conference, the reasons for transfer to the skilled nursing facility and plans for followup were presented to the patient's parents. They were angry and disappointed, but reluctantly accepted the decision. The social worker would continue to communicate with them regarding the ongoing involvement of the rehabilitation medicine service, and the physiatrist and rehabilitation nurse specialist would be available to talk with them. They were invited to accompany the patient to his rehabilitation medicine as well as to his other clinic followup appointments.

During the next 4 months, the patient slowly became less agitated and more cooperative. He began to follow commands and attempted communication. Speech therapy was increased to 5 days each week and a barium swallow was done. This showed aspiration of thick and thin liquids, but not solids. The speech therapist began working with him on swallowing as well as on communication. At the monthly interdisciplinary team conference, it was determined that the patient was ready for another trial of comprehensive rehabilitation, and plans for transfer back to the rehabilitation unit were made.

At readmission to the rehabilitation unit, the patient was awake and alert and followed simple, one-step commands. He could verbalize with his tracheostomy occluded, but speech was dysarthric, low volume, and confused. He also had significant motor and verbal perseveration.

He now had voluntary, isolated movement in the left lower extremity more than in the left upper extremity. Right upper and lower extremity strength was normal. Static sitting balance was good, bed mobility was independent, and transfers required minimal assistance by one person. Static standing balance was poor, primarily due to ataxia, and he ambulated only three to five steps with the assistance of two people. He was now continent of urine and on a voiding schedule every 4 hours, and was continent of bowel without suppositories. He was eating solids and taking thickened liquids, but only when under the direct supervision of the speech therapist. Body weight and serum chemistries were normal. Tracheostomy and gastrostomy tubes were still in place. He remained on carbamazepine and amitriptyline and had not required medication for agitation for the past 2 months.

The patient was referred to physical therapy for continued active range-of-motion, and progressive resistive and endurance exercise activities. The occupational therapist was consulted for perceptual and cognitive evaluation and remediation, upper extremity evaluation, and ADL evaluation and training. The speech therapist was consulted to continue work on swallowing and language and communication skills. A repeat barium swallow was also ordered. The psychologist was consulted for neuropsychiatric evaluation and testing. The social worker was to continue working with the family and patient on discharge plans, while the plastic surgeon was consulted for follow-up of plans for facial reconstruction.

The first team conference was held 10 days after readmission to the rehabilitation unit, and the physiatrist reported that the plastic surgeon wanted to schedule the first phase of the facial reconstruction within the next 4 weeks. The patient's parents were still very anxious for this surgery; however, the physiatrist was concerned that exposure to general anesthesia and the stress of surgery would compromise the cognitive recovery that was occurring. The family decided to go ahead with the surgery.

The physical therapist reported that the patient now had 4/5 strength throughout the left lower extremity, good dynamic sitting balance, standing balance with supervision, and verbal cues. He was able to transfer with supervision and verbal cues, and ambulate for 20 ft. with minimal to moderate assistance by two people. Upper and lower extremity joint range-of-motion was within normal limits. The patient still required constant cueing to perform his exercise program and exhibited little carryover of learning. The occupational therapists reported that there was no apparent left side neglect, but there was evidence of severe learning and memory deficits. The patient also exhibited components of motor apraxia in both upper extremities. He was capable of light hygiene activities, but required constant cues to complete tasks.

The speech therapist reported that a repeat barium swallow had shown minimal aspiration of thin liquids only and the patient had been placed on a mechanical soft diet with thickened liquids, which he was tolerating well. His speech showed components of both dysarthria and confused language, but no significant elements of aphasia had been identified. The rehabilitation nursing staff reported that the patient was continent during the day, but had occasional accidents at night. Although he at times became confused, he was not combative. He required constant reminders and cueing during functional activities and sometimes claimed he had not received his meals when he had eaten everything on the tray. The dietitian reported that the patient was receiving adequate nutrition by mouth, and tube feedings had been discontinued. The psychologist reported that testing was very slow due to limited attention span and poor carryover of information. Severe deficits in ability to form new memories and in information processing had been uncovered. Reading comprehension was also limited, but this may have been preexistent to the brain injury.

The social worker reported that the patient's family would not care for him or take him home unless he was "normal." They expressed the opinion that it was the government's responsibility to care for him. They had, however, sought and received legal guardianship and were managing his financial affairs. The social worker had also determined that the patient was still on active duty military status. The military healthcare facility where he was first treated was requesting updated information for completion of the MEB.

New goals were set, including improved ADL with supervision and visual cues (pictures, simple lists), use of an activity logbook to aid memory, independence in the exercise program with visual cues, ambulation with contact guard assistance of one or two people and nighttime continence. The physiatrist would confirm the surgery date and prepare a summary for the patient's military physician. It was also decided to start tapering the amitriptyline since agitation was no longer a problem. The social worker would investigate alternatives to his discharge home.

The first phase of the patient's facial reconstruction was scheduled for 3 weeks later. Two weeks after the initial team conference, the physical therapist reported that the patient was ambulating with contact guard assistance of one person due to occasional loss of balance. He could also ascend and descend a flight of stairs with one railing and contact guard assistance. He continued to require verbal cues to complete his exercise program, but was improving. The occupational therapist reported the patient could complete simple hygiene tasks with setup and occasional cues. He required maximal assistance to make entries in his activities logbook and did not spontaneously use the book to assist his memory. He was working on dexterity activities and showing steady improvement. The speech and language department reported that verbal output was more comprehensible and appropriate, and that he had no dietary restrictions at present. The psychologist reported that his memory and learning skills remained poor, but his ability to comprehend written information was slowly improving. The social worker reported that the patient's family was more content now that the facial surgery was scheduled and the patient was showing some improvement. However, they were becoming less available, that is, visiting only once or twice a week for short periods and not promptly returning phone calls. The physiatrist reported that the patient would be transferred to plastic surgery service preoperatively and would remain there postoperatively until he was medically stable.

The patient underwent facial surgery. Postoperatively, he required heavy sedation to control pain. Rehabilitation medicine consultation service followed the patient on the surgical ward and recommended restarting tube feeding to preserve nutritional status. They also recommended that when the patient was alert, he should resume physical, occupational, and speech therapy as tolerated. Unfortunately, the patient's postoperative course was complicated by fever and diffuse infection of the frontal bone flap. The patient required aggressive fever management and removal of the frontal bone flap. Treatment with intravenous antibiotics was recommended for at least 6 weeks. After 1 week of antibiotic therapy, the patient was able to resume rehabilitation therapies on a limited basis. At 3 weeks postoperatively, he was participating in a full rehabilitation program and returned to the rehabilitation medicine unit with a Hickman catheter in place. Due to the infection, no further facial reconstruction was planned for at least 6 months.

The patient continued physical therapy, occupational therapy, speech therapy, and meeting with the psychologist. Every other week, goals were set at the team conference. Six weeks after his return to the rehabilitation unit, the patient was independent in ambulating, performing a resistive exercise program, and in ADL. However, his memory and ability to use assistive devices, such as the activities logbook, remained poor. Due to the memory deficits and inability to learn new information, it was determined that the patient would require a closely supervised living situation. The social worker had learned that he had been placed on the Temporary Disabled Retirement List by the military, making him ineligible for VA vocational services. His family was still unable to care for him at home. Placement in a VA domiciliary care facility was initiated, and the patient was transferred to the domiciliary care facility 2 weeks later.

Followup in the Rehabilitation Medicine Service clinic continued at 3-month intervals. The patient's memory and cognitive deficits showed minor improvements as he acclimatized to his surroundings. He received a medical discharge from the military with 100% disability, and it was recommended that he be referred to VA vocational rehabilitation for evaluation for independent living support services and possibly sheltered employment. The patient's family, however, refused this until facial reconstruction was complete. Since his parents were his legal guardians, no further action could be taken. After 1 year, he was discharged from the rehabilitation medicine clinic. He continues to undergo staged reconstruction of his face and resides in the domiciliary care facility.

Comment

Traumatic brain injury without other complicating injuries is rare, and the care of these multiple-injured patients requires the coordination of many medical and surgical specialists. To avoid conflicting medical and surgical treatment plans, it is essential that one physician review all the recommendations, resolve conflicts, and assure that all issues are addressed. A physiatrist is uniquely suited to this role.

In addition, traumatic brain injury has effects that reach far beyond the injured individual to his family, friends, and community. Unfortunately, return to an independent lifestyle is not always possible, and alternative, appropriate living arrangements must be made. A major role of the rehabilitation team is to assist the family in recognizing the deficits that will prevent return to a "normal" life and to set appropriate goals and make plans. Family members often have difficulty accepting the changes in personality and behavior that result from traumatic brain injury. Unless the interdisciplinary team members have communicated effectively among themselves and arrived at a consensus plan and goals, conflicting information and attitudes may reinforce the family's denial, impairing the ability to move forward with disposition planning.

Close followup after the patient's transfer to a skilled care facility allows for prevention of secondary complications, such as joint flexion contractures and skin breakdown, as well as the recognition of spontaneous recovery and the appropriateness of different levels of rehabilitation services.

Case Study 4: Spinal Cord Injury

A 20-year-old soldier sustained a thoracic vertebrae fracture/dislocation at T-9 and T-10 in a two-car, head-on collision. At the scene, the patient reported his legs were numb and he could not move them. He was placed on a back board and transported to a local hospital. Initial evaluation revealed stable vital signs, normal level of consciousness and normal upper extremity strength, but no sensation below the level of umbilicus, and no volitional movement of the lower extremities or lower quadrant abdominal muscles. Radiographs of the cervical spine showed no fracture or instability. Thoracolumbar spine radiographs showed complete anterior dislocation of T-9 on T-10. Foley catheterization of the bladder yielded 1,000 mL of clear urine. A CT scan of the fracture area showed multiple fragments and complete disruption of the spinal canal and its contents. The patient was placed on a Stryker frame and admitted to orthopedic surgery service.

Ten days elapsed before his transfer to a VA Spinal Cord Injury Center could be arranged. On arrival at the spinal cord injury center, the patient was found to have motor and sensory complete T-10 paraplegia. A Foley catheter was in place and drained cloudy, foul smelling urine. Rectal examination was consistent with fecal impaction. A grade I decubitus ulcer had formed over the sacral area and large blisters over both heels. Joint range-of-motion at the hips showed mild limitations of internal rotation, and ankles dorsiflexed only to neutral. Tone was returning to the lower extremities and the patient reported occasional spasms. He was placed in a Gutmann bed with orders for position change from side to side every 2 hours and no supine positioning. Urine was sent for analysis and culture. The Foley catheter was removed and an every 6-hour intermittent catheterization regimen begun. Digital disimpaction and enemas were ordered; a bowel program consisting of high fiber diet, stool softener, and every-other-day suppositories was initiated.

Deep vein thrombosis prophylaxis was ordered consisting of 5,000 units of subcutaneous heparin 3 times a day. Orthopedic spine surgery service was consulted for management of the thoracic fracture/dislocation. The patient was referred to physical therapy for resistive exercise of the upper extremities and passive range-of-motion of the hips, knees, and ankles at bedside. The occupational therapist was consulted for foot-drop splints and assistive devices for ADL. The psychologist was consulted to provide emotional support and counseling. The social worker began investigation of the patient's social situation and resources, and acted as liaison among the patient, the family, and the military administration. The recreational therapy department was asked to explore the patient's avocational interests and activities.

At the team conference 1 week after admission, the physiatrist reported that the patient's fracture was judged stable and he had been cleared to get out of bed. An abdominal binder was used to provide increased ventilation, secretion clearance, and trunk support. Urine culture was positive for infection, and the patient was being treated with oral antibiotics. The rehabilitation nursing staff reported that the patient was cooperative and pleasant. His bowel program was effective and bladder volumes were under 400 mL. There had been no spontaneous voiding. The sacral area was completely healed. The blisters over the heels had been unroofed and there was healthy granulation tissue at the bases. The patient was tolerating the Gutmann bed well.

The physical therapist reported that the patient was maintaining normal upper extremity strength through Theraband and free weight exercises. He was independent in his exercise program. His lower extremity range-of-motion at the hips, knees, and ankles was now normal. Tone in the lower extremities was increasing bilaterally with several beats of unsustained clonus at the ankles. Frequency of spasms was increasing, but these were not currently interfering with function. The occupational therapist reported that evaluation of the patient's ADL abilities had been limited by the patient having to remain flat in bed. There were no deficits in dexterity identified at this time, and occupational therapy was on hold until the patient could be mobilized. The psychologist reported that the patient's spirits were good, but there appeared to be a significant degree of denial regarding his injury and potential recovery. The social worker reported that the patient was single and had been living in the barracks. He had been raised in a single parent home and that parent currently could not be located. He had a GED (General Education Diploma) and had been an airborne ranger. At the time of the injury, he had been training for a triathlon. The recreational therapist reported that all the patient's avocational interests had centered around sports, both as participant and spectator. At present, he seemed to be content to watch television and wait until he "got better." The team concurred that the patient was obviously in a phase of denial and, given his past emphasis on physical activities, adjustment to his impairments would be difficult. All team members would gently but firmly reinforce that recovery of lower extremity function was unlikely while stressing the patient's residual ability and potential for participation in adapted activities and sports.

The treatment plan was to transfer the patient into a regular hospital bed and to begin working with him to become more mobile. An abdominal binder was ordered. Goals for the next 2 weeks were set at independent bed mobility, tolerance of sitting for 2 hours at a time, fitting of an appropriate wheelchair, independence in upper body hygiene, feeding and grooming activities, continued control of bowel and bladder, and healing of the heel ulcers.

Two weeks later the rehabilitation nurse reported that the patient had several episodes of spontaneous voiding. Postvoid residual urine volume was 200-300 mL. The ulcers over the heels were nearly healed. He had, however, developed a right, grade I ischial ulcer after sitting in his wheelchair for 2.5 hours one day. His wheelchair cushion was changed, and the nursing staff reinforced the need for pressure releases every 5 to 10 minutes. The physical therapist reported that the patient was independent in bed mobility. He had had no problem tolerating the upright position and was able to maintain sitting on the edge of the exercise mat with upper extremity support. He required the moderate assistance of one person to go from supine to sitting and the moderate assistance of two people for sliding board transfers. He could propel his wheelchair for unlimited distances. Joint range-of-motion was being maintained throughout the lower extremities, and the patient required only occasional assistance and cueing with his range-of-motion program.

The occupational therapist reported that the patient was independent in feeding, upper body hygiene, and dressing with set up. He required minimal assistance and adaptive devices for lower extremity dressing. The psychologist reported that his spirits remained good, though there was still concern about denial. Socialization with the other patients on the unit was being encouraged, and the recreational therapist reported that the patient was working on craft activities in a group setting. He had not yet participated in any outings. The social worker reported that the patient's MEB had not yet been done and the army was requesting an updated medical summary.

It was agreed that during the next 2 weeks the physiatrist would perform urodynamic studies of the patient's bladder and change his bladder program if needed. The physiatrist would also provide the military with an updated medical summary. The physical therapist's goal would be sliding board transfers with minimal assistance and independent supine to sit transfers using an overhead trapeze. Need for pressure release when sitting would be stressed. The occupational therapist would begin working on bathroom transfers and activities. The psychologist would continue to provide support while the recreational therapy department would include the patient in the next outing.

At the next team conference 2 weeks later, the physiatrist reported that urodynamic testing had shown a spastic bladder with a significant degree of dyssynergia and vesicoureteral reflux. The intermittent catheterization program would be continued. The nursing staff reported that the patient had been compliant with his skin care regimen and there were no new skin problems. The physical therapist reported that spasticity was becoming more of a problem, and spasms were interfering with transfer. At this point the patient could transfer with the minimal assistance of one person, but as a safety precaution, a second person needed to be available. It was also noted that left hip range-of-motion, especially rotation, had become limited. The occupational therapist concurred that spasticity was interfering with bathroom transfers. The recreational therapist reported that the patient had gone out to the movies, but that he had appeared somewhat self-conscious and frustrated at his dependence. The psychologist reported that the patient was less cheerful but had not yet started talking about his injury and disabilities. The nursing staff added that a few of his friends from high school had come to visit over the weekend and, although he had enjoyed the visit, he was withdrawn afterwards.

The possibility of heterotopic ossification at the left hip was discussed. The physiatrist would order plain radiographs and, if these were normal, a bone scan would be done. Urinary tract infection was also considered as an etiology of increased spasticity and a urine culture was ordered. The physical therapist would continue to work on transfer skills and would monitor more closely the patient's range-of-motion program. The occupational therapist would continue the current treatment plan, but would begin some prevocational testing and discussions with the patient. The recreational therapist would continue to include the patient in activities and try to arrange for him to attend a wheelchair basketball game. The psychologist would continue to provide support and counseling.

Radiographs of the left hip were normal, but bone scan showed abnormality at the left hip consistent with heterotopic bone formation. The patient was started on etidronate disodium (Didronel). Urine culture was positive for infection and an appropriate antibiotic regimen was begun. Within a few days of treatment initiation, spasticity was less marked and not interfering with activity. At the next team conference, the physical therapist reported that the patient required supervision only with occasional contact guard assistance for all transfers. Left hip range-of-motion was still decreased, but improving. The occupational therapist reported that the patient was independent in self-care activities but had been resistant to prevocational assessment and discussion. The recreational therapist reported that the patient had attended one basketball game, but declined to attend a second. While at the game, he had been critical of the activity. The psychologist reported that the patient was beginning to recognize the permanence of his impairments and the changes to his lifestyle, and the rehabilitation nursing staff reported that the patient had begun talking to one of the young, male, night nursing aides about his disability. The social worker reported that neither the patient's MEB nor PEB had been completed.

The plan for the next 2 weeks was to continue working on functional and self-care abilities. The occupational therapist would continue with the prevocational evaluation, and the patient would also be referred for handicapped driver-training assessment. Because of special regulations for spinal cord injured service members, this patient was eligible for VA vocational rehabilitation counseling services before his discharge from the military.

During the next 3 months the patient continued to progress in all areas of independent functioning. He went through a period of depression and anger about his injury and impairments but, with the support of the team and other patients, was able to come through this and begin planning his future. This included counseling regarding sexual function and fertility. With the aide of a social worker he was able to find an apartment near his old neighborhood and to purchase a car. He decided to pursue training as a photographer and would begin classes in the fall. After 4 months in the rehabilitation unit he was discharged to live independently. He continued to receive his healthcare at the VA spinal cord center as an outpatient.

Comment

As a patient moves through a rehabilitation program, his needs change. In response to these changing needs, the composition of the interdisciplinary team must change. In the case of a spinal cord injured patient, the initial area of concentration is on physical abilities and medical management, but as the patient learns new skills, the emphasis shifts to social and vocational issues.

Case Study 5: Cervical Strain

A 37-year-old supply sergeant experienced neck pain and stiffness after a box of boots fell from the top of a stack and struck him on the side of his head. There was no loss of consciousness. He was initially seen in the troop medical clinic by the general medical officer (GMO) and complained of occasional electric shock-like pain radiating down his right arm and paresthesias in the thumb and index finger when lying supine or turning his head to the right. A nonsteroidal antiinflammatory drug (NSAID), a soft cervical collar, and physical therapy were ordered.

A physical therapist's evaluation of the patient revealed paraspinal muscle spasm with decreased neck rotation and lateral flexion. The right brachioradialis muscle stretch reflex was decreased compared to the left and there was mild weakness of the wrist extensors, forearm pronation and elbow flexion on the right. The physical therapist contacted the GMO to discuss the neurologic findings and proposed treatment plan.

After discussion, cervical spine radiographs were ordered to include flexion/extension views. These showed no fracture or instability. A treatment program including cervical traction, superficial heat, NSAID, active cervical and shoulder range-of-motion, and a Philadelphia collar was developed. Reevaluation of the patient's neurological status was done daily by the physical therapist.

After 2 weeks, the patient reported a 75% decrease in neck pain, occasional radicular pain, and resolution of the paresthesias. Neck range-of-motion was still limited, and cervical paraspinal spasm was still evident but decreased. Strength in the right upper extremity was stable as were muscle stretch reflexes. The GMO confirmed the therapist's findings and the treatment regimen was continued.

After 4 weeks of treatment, the patient reported complete resolution of the radicular pain and about a 90% decrease in neck pain. Muscle stretch reflexes were now symmetric, but the right arm remained slightly weak. Cervical spine range-of-motion was now normal and spasm was resolved. After discussion, cervical traction was discontinued and neck range-of-motion against gentle resistance was begun. Progressive resistive exercises for the right upper extremity were also begun. The GMO provided the patient with a temporary profile excusing him from running, lifting more than 10 lb, overhead work, and pushups for the next 2 months; however, the patient was instructed to use the stationary bicycles and stair climbers in the base gymnasium to maintain aerobic conditioning. Over the next month the patient continued to be seen in physical therapy at increasing intervals. His upper extremity strengthening program was transferred to the base gymnasium, and he was instructed to gradually resume other activities. Two months after his initial injury he was discharged from physical therapy and returned to full duty.

Comment

This last case illustrates that interdisciplinary management does not take place only in the physiatry clinic, but can be accomplished at the troop medical clinic level. The interdisciplinary management of this case differed from the more common multidisciplinary management because of the communication between the physician and the therapist, and the formation of an integrated treatment plan, including pharmacologic intervention, restriction of activity, and exercise and physical modalities. Without effective communication between the therapist and physician, inappropriate treatment may have led to further or prolonged impairment. Also important is the attention to the patient's overall health and fitness. If the patient had simply been given a profile but not instructed in alternative ways to maintain his aerobic conditioning, he would have required further rehabilitation once his return to normal activity was allowed. Even worse, he may have suffered secondary injury due to deconditioning when he returned to full activity.

In summation, the five previous cases highlight the importance of involvement of the patient as well as any other involved family members or caregivers in the rehabilitation process. It has been pointed out that the patient is the only one who is intricately involved in all phases of the rehabilitation program. Furthermore, as the patient moves through the rehabilitation process, team members may change, but the foundation of interdisciplinary management and communication remains the same.

CONCLUSION

The rehabilitation of transient or permanent physical impairment requires the cooperation and work of a wide variety of individual healthcare practitioners. Whether the impairment is relatively simple and temporary (such as the case of a distal femoral fracture, Case Study 1), or complex and permanent (in the case of severe traumatic brain injury, Case Study 3), management by an interdisciplinary team will clarify goals, coordinate treatment plans, reduce the redundancy of efforts, and lessen or help to avoid the impact of secondary complications.

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Chapter 15

VOCATIONAL REHABILITATION AND COMMUNITY REINTEGRATION OF THE WOUNDED COMBATANT

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INTRODUCTION

IN-HOSPITAL PHASE FOR THE INJURED SOLDIER
- **Emotional Reaction to War-Related Injuries**
- **Short-Term Counseling**
- **Vocational Assessment**

POSTHOSPITAL PHASE

CONCLUSION

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INTRODUCTION

Though the collapse of communism indicates a diminishing probability that the United States will be engaged in global wars, there is still the possibility of limited conflicts involving U.S. troops. Any military action, of course, brings casualties, and these injuries have immense personal, social, and economic consequences. Moreover, even in peacetime, military personnel may suffer service-connected disabling injuries. One implication of traumatic injuries that is beginning to receive attention from healthcare providers is the manner in which an effective vocational rehabilitation process can be used to facilitate the soldier's return to duty, or assist in optimal life adjustment when resumption of military service is not possible.

This chapter will identify and discuss the different components or steps for an effective vocational rehabilitation process. The context for this discussion will be the two phases of vocational rehabilitation, namely, the in-hospital and outpatient/post-discharge periods. Planning for different career options should begin as soon as possible after the soldier's injury. Early intervention can assist the individual in making a personal commitment to reestablish a sense of order in his life; the person's level of motivation to recover can be stimulated and maintained more effectively than if sick-role behavior is allowed to establish itself; and early availability of rehabilitation opportunities can ease the process of coping and adjustment.[1]

IN-HOSPITAL PHASE FOR THE INJURED SOLDIER

If vocational rehabilitation is to benefit the wounded soldier, three areas must receive attention as the soldier receives in-hospital medical treatment. These areas are: (1) recognition of the individual's emotional reaction to the injury, (2) short-term counseling that addresses emotional and related career-adjustment issues, and (3) vocational assessment. Many disabilities may not stabilize sufficiently during the in-hospital course to permit a completely accurate assessment of future capabilities and opportunities. Nevertheless, vocational evaluation initiated within the hospital can still assist the soldier in learning about posthospital discharge career possibilities.

Emotional Reaction to War-Related Injuries

Determinants of Emotional Reaction

Battle wounds not only inflict damage to a person's body; they also affect an individual's emotional life. To neglect what is happening emotionally to a soldier after an injury can considerably endanger the timely and appropriate development of vocational rehabilitation plans. There is frequently a close relationship of the physical condition to the psychological condition, and there is evidence that addressing psychological issues can speed physical recovery.[2,3] A war-related injury has important psychosocial dynamics that are distinguishable from the psychosocial dynamics of other disabilities of similar sudden onset.

These dynamics can consist of (*a*) the military service's expectations that one will make every effort, when appropriate, to return to duty; (*b*) the dependence factor that may be integral to a large part of the person's military life before injury onset, a dependence that was fostered to ensure that orders would be obeyed promptly; and (*c*) expressed feelings of courage that may mask a denial of the injury's implications for independent living. Courage is earnestly promoted as an important characteristic of a soldier, and is conceptualized as perseverance in the face of recognized difficulties. All three of these dynamics can influence the soldier's emotional adjustment to bodily injuries and losses incurred during war. Expectations required during military service may exacerbate the individual's anxiety and fear that he may not again be an effective soldier, dependence may inhibit the assumption of personal responsibility for one's career outcomes, and denial with the mask of courage may cause the soldier to ignore important health measures and vocational rehabilitation interventions.

How the individual responds emotionally to the injury can also depend on the following factors:

- The nature of the injury, especially its visibility and severity.
- The age of the person.
- The degree of satisfaction with military life prior to being wounded.
- The person's level of dependency needs as well as his philosophy of life.

- Whether early counseling or vocational rehabilitation or both occurred.
- The circumstance of injury onset (whether the injury happened on the battlefield or in a support unit).

Each, or a combination of these factors, may influence the person's postinjury behavior. For example, if the soldier is accustomed to being dependent on others for most daily needs and has always been reluctant to show initiative or independent behavior, then he may react to the threat of disability by increasing this prewound mode of behavior.

The nature of the injury, such as those that are disfiguring, disabling, painful, or on a body region that carries special importance, like the eyes or reproductive organs, may have a psychological significance that has little to do with biological factors related to survival.[4] If the individual's military responsibilities provided continued dissatisfaction, then the person may find in the injury an excuse not to return to active duty. Finally, the age of the wounded soldier can have a decided impact on motivation and even on the level of willingness to appreciate possible benefits accruing from return to duty. A person with many years of service who is looking forward to retirement usually has different concerns than a soldier early in his career. The latter, while coping with young adult issues, may have a different perception of injury-related implications.

Emotional Reactions and Needs

Following the injury, the wounded person may have a variety of different emotional reactions, such as anger and hostility, frustration, feelings of damage and powerlessness, fear of the unknown future, depression, and perhaps guilt. Identifying these distinct emotions helps in understanding the person's response to war-related injuries. Another approach is to view the wounded soldier as undergoing sequential, reactive stages to the unexpected event. The stages in the process of adjustment to disability are usually transitional and temporary in nature, and the stages themselves are not necessarily discrete and categorically exclusive. They may fluctuate, blend, or overlap with one another.[5]

Roessler[6] has conceptualized that successful adaptation to a disability is the result of two adjustment processes: acknowledgment and goal setting. Acknowledgment encompasses two steps, *appraisal* and *reappraisal*. Appraisal consists of an immediate positive or negative affective reaction, followed by a cognitive evaluation in which the individual assesses the seriousness of the event, its impact on return to military life, and the immediate ratio of personal deficits to personal resources. Reappraisal involves the injured person in such mental processes as the consideration of self beliefs, values, expectations, and perceived incentives and disincentives.[6] If the individual is going to adapt successfully to the injury, then the injury event must be interpreted using cognitions associated with internal control and positive self-esteem. Finally, goalsetting involves *remotivation* and *restructuring* processes that include the use of such positive coping mechanisms as (*a*) neutralizing the perceived negative implications by controlling the meaning of the event; (*b*) minimizing personal discomfort; or (*c*) using the problem-solving behaviors of generating alternatives, selecting a solution, and monitoring the outcomes of a selected strategy that leads to return to duty or a service discharge with optimal utilization of residual capacities in civilian employment.[6]

This cycle of appraisal, reappraisal, remotivation, and restructuring assumes that the usual adaptation to a disability involves a temporal sequence of psychosocial developmental stages.[5] The cycle also provides an understanding of how someone wounded in combat can achieve successful adaptation, and it identifies the specific needs that an individual may have during the in-hospital recuperative or rehabilitation periods or both. For example, Table 15-1[5] illustrates the relationship between the particular cycle of the individual's response and selected, emerging needs.

A wounded soldier's emotional reaction to an injury can be understood, consequently, as a cycle of stages during which individual needs, coping styles, and dominant emotions emerge. These needs and emotions can become the focus of initial rehabilitation efforts. This is illustrated in the following case study.

Case Study 1

Corporal Anderson was injured during the Vietnam War. When he was blown off a bridge by a mortar shell and landed in a gully, he incurred shrapnel wounds in his back. During the in-hospital phase of his treatment, he began to consider his career options. He was very upset that he might not be able to return to duty because of the severity of his injury. Anger and frustration were the dominant emotions at this time, which he showed in frequent, verbal outbursts to the healthcare staff. But as he learned more about the effects of his injury (fragments had caused

TABLE 15-1

ADAPTIVE STAGES TO A DISABILITY EVENT[5]

Stage	Need
Appraisal	To obtain information about career options To receive emotional support To be acknowledged as a worthwhile person
Reappraisal	To obtain information about residual assets and social and vocational role opportunities To assume personal responsibility To identify new life goals To accept others and their support
Remotivation	To receive information about effective coping strategies To develop career goals that include realistic options To receive support To review personal resources and responses and how they can be used in expected, stressful situations
Restructuring	To identify stressful situations related to implementing goals To identify additional, personal skills needed to reach goals To implement useful coping strategies To receive support

severe nerve damage, resulting in difficulty for prolonged standing or sitting), he began to consider new career goals. This process was nurtured by continued information about career opportunities, the support of his family who was pleased that he had survived the war and was returning home, and by his physicians who continually stressed new, challenging opportunities. The corporal's anger and frustration began to subside, and the appraisal and reappraisal stages progressed to a remotivation period. This stage was characterized by a review of his personal resources during which he gradually acknowledged his limitations and strengths. Anger continued over his situation but with an absence of verbal outbursts, and a formulation of new career goals was achieved.

A key point in this soldier's transition to the remotivation stage was early intervention in regard to his emotional concerns, particularly to his feelings of uncertainty and frustration about the future. With wounded soldiers, early attention to these concerns can make a decided difference in whether the person will have, during hospitalization, a positive or negative orientation to the resumption, when possible, of military responsibilities.

Short-Term Counseling

The onset of a war-related injury can lead to many dramatic changes in a person's life. Not all of these changes may be immediately apparent; some present themselves to a person and to health-care providers over a long period of time. Some of the immediate changes include (*a*) adjusting to an awareness of new limitations of body or mind, (*b*) learning how to deal with a possible modification in career plans, and (*c*) engaging in a resocialization process when friends or family relate to the person in a different way, especially before the injury has stabilized and more definite life plans can be formulated. In-hospital rehabilitation then becomes a process of assisting the individual to live with the injury in the hospital environment, and helping him to prepare to follow a career option after gaining information about possible opportunities.

Counseling is part of this dynamic process of learning and exploring new career directions. The goals of counseling are many and include the following:

- Assisting the person to understand the emotional reactions to the injury and to learn how to deal with any negative reactions caused by personal frustrations or the attitudes of others.
- Identifying personal resources, such as educational background, work experience, and definite occupational interests that can be

developed for vocational opportunities, either with the option of return to duty or of civilian adjustment, including employment.
- Providing support to the individual as adjustment attempts are made during hospitalization and vocational plans are developed.
- Educating the person with sudden-onset disability to develop effective coping mechanisms and to set appropriate priorities for each. These include the use of assertive responses; relaxation procedures; redefinition of personal, social, and vocational goals; and problem-solving techniques.
- Ameliorating feelings of alienation and isolation, and modifying denial beliefs that would hinder rehabilitation efforts.

To achieve these goals, counseling for the individual involves communicating information with respect and support, and educating the person to appreciate his capabilities. This understanding can stimulate feelings of responsibility, optimism, and worth. Counseling may also demand periodic confrontation when the soldier is pressured with diverse, but necessary, options and urged to explore each direction carefully.

These supportive efforts, however, should be directed toward working within the person's lifestyle, and must include an awareness of the cultural values of the soldier and the impact of these values on career choices.[7] The armed services represent many different ethnic and cultural groups, and some members of these groups may be less comfortable than others with the traditional counseling approaches that emphasize verbal interaction and disclosure of emotionally loaded personal information.[8] The modes of interaction and patterns of authority, decision-making processes, and roles of military service personnel are in large part determined by ethnic and military cultures. Counseling strategies that recognize the importance of differences generated by both military life and ethnicity provide valuable support for the development of intervention strategies built on patterns familiar to the individual. For example, research[9,10] has suggested that as a group, Hispanics and African-Americans who are in counseling tend to be less introspective, less introverted, and less concerned with self than what is considered the cultural norm for middle-class white Americans on whom traditional counseling approaches were developed. Substantial differences also exist in attitudes, expectations, and general lifestyles, and the counselor must be prepared to deal with them.[11]

If such conclusions are applicable to today's soldiers from African-American or Hispanic backgrounds, more appropriate counseling strategies should be used, that is, those that (*a*) do not ask for extensive self-disclosures, (*b*) are short term, and (*c*) emphasize communication that is related to the person's current experiences and practical options.[12–14] The use of concepts such as "self-esteem" or "self-insight" is minimized during counseling interactions. The dialogue concentrates more on what is happening to the individual, the personal meaning of what has occurred, and understanding what the future offers. Counseling effectiveness with soldiers representing different racial and ethnic backgrounds depends, however, on the type of presenting problem and characteristics of the counselor that may override racial and ethnic differences.[11]

To maintain a supportive approach that recognizes the importance of the individual's lifestyle, the most promising medium for communicating may be group counseling. Remotivation and self-understanding can often occur because of the influence of one's peers. Through the facilitation of the group leader, the injured person's peers may be able to communicate that it is permissible to feel angry, frustrated, and even guilty over what has happened, and that one can profit by mistakes. Military personnel, particularly those who experience injury in a noncombat position, may blame themselves for the casualty. Yet in a group counseling setting, where opinions are respected and feelings shared, the individual may gain another perspective on his beliefs and perhaps acquire new convictions that there are realistic, attractive career options.

Sharing information, active listening, and showing support can all assist the individual in awareness of personal resources, varied obstacles, modified life goals, and effective coping strategies to deal with war-injury implications. In turn, understanding and utilization of coping methods assist the person to become an invaluable partner with the healthcare provider for the achievement of rehabilitation goals. This partnership is absolutely necessary if the soldier accepts the implications of the injury and is to have energy to take responsibility for tasks directed either to the return to duty or to military discharge with optimal career placement. As stated earlier, a war-related injury brings personal loss, a loss that can be minimized by the sharing of the experience, the opportunity to talk about and work through the sense of loss, attention to personal strengths, and a realistic projection to the future.

Even though counseling may be designed to meet individual needs, resistance from the injured person may be a dominant factor during hospitalization. A reluctance to explore career options, or to respond to other rehabilitation interventions should be identified as soon as possible. Frequently, this reluctance to any counseling or vocation assessment is to be expected. Someone may require a period of time to work through feelings associated with the injury event. During this time, support in the form of listening and showing respect and acceptance of a person's emotions is necessary. If reluctance for any rehabilitation assistance continues during the later phases of the person's in-hospital recovery period, the counselor should identify the cause of the reluctance, convey to the person that he is aware of the reluctance and its cause, and then develop a strategy to deal with the resistance. An example of reluctance is illustrated in the following case example.

Case Study 2

Specialist Smith had been a robust, athletic army medic until he stepped on a land mine during combat. He was lucky to be alive, and to have had only a below-the-knee amputation. But his physical system had undergone a severe insult, and the surgery to reconstruct his stump had been a nightmare of pain and sleepless nights. He had served as a medic for 3 years. Prior to entering the military service, he had graduated from high school and earned an Associate of Arts degree as a veterinarian assistant from a community college. Personal history indicated that he was well liked among his service peers, had advanced quickly in rank, and was considered by the doctors to be a "top-flight" medic.

Specialist Smith had lived on a farm before his military service. Although he believed that his combat injuries would not leave him totally incapacitated, he felt different about himself and seemed depressed and distracted. The nurses recognized that it was not unusual to be depressed, but sensed that Smith's reaction was more pervasive than was typically the case. He frequently yelled at any nurse, doctor, medic, or ambulatory patient he could see. He ate little and refused almost anything except the ice cream sodas his doting mother brought every day. In the doctor's mind, Smith seemed to be questioning his identity and showed an inability to say what kind of a person he was prior to the injury event. During an initial visit with a counselor, he revealed that there was a strong parental expectation that he take over the family farm after his father was too old to work it. He had acquiesced to this expectation, which was largely unspoken but assumed, because he had great difficulty in communicating with his parents. Before his hospitalization he had learned to deny his feelings, but the counselor surmised that there was an apparent naiveté about the world, his career options, and what was required to achieve a realistic, vocational goal. He did not wish to discuss much about his life with a counselor or related healthcare provider, and remarked to a patient that "I don't know what to do. Before this happened, I had planned to stay in the service. I like to work with animals, but I don't want to be a farmer."

To deal with Smith's apparent reluctance for counseling intervention and exploration of career options, the counselor should convey support for Smith's dilemma, and give a listening ear to the different life and career-related issues. The mother, as a possible reinforcement for the son's current acting-out behavior, must also be monitored and redirected. Group counseling should also be considered because his peers might have an influence on minimizing the angry behaviors. If the counselor shows respect for Smith's career dilemma, and understands and shares this knowledge of how the recent combat injury might be aggravating the dilemma, this acceptance could convey the important message: "You are not alone. There are attractive options, and there are both your own and environmental resources to assist you." The availability of additional data on career alternatives and new information about his remaining functional capacities will assist Specialist Smith in his reappraisal of the injury event. In addition, if the counselor helps him to become aware of the core problem related to his current behavior and teaches him problem-solving skills that can alleviate the personal difficulties associated with decision making, this assistance can facilitate Smith's own remotivation to career and life adjustment goals.

Vocational Assessment

While the soldier is undergoing in-hospital rehabilitation and treatment, this third area of intervention effort could be considered the most important. If the individual receives feedback about remaining postinjury strengths (early in the recuperative process), this knowledge may stimulate a motivation to recover. The process of adjustment to a disruptive life event has been extensively studied by social scientists.[1] The preferred mode of intervention with recently injured persons is a highly individualized and flexible approach characterized by early assistance for any possible alterations of cognition that may be harmful to the mental health and career adjustment of the soldier, and by extreme sensitivity to the particular emotions that each wounded person is experiencing.[5] Knowledge acquired from an individualized assessment approach can help the solider to counteract feelings of frustration, guilt, and even hopelessness over return to duty or, if that option is not possible, over a satisfying, civilian job placement.

Because rehabilitation for the injured soldier is a process of restoration—a way to help an individual toward practical goals where opportunities for self-dependence and personal satisfaction are possible—it is a process based on productive output and productive living. Vocational assessment is then a comprehensive, interdisciplinary process of evaluating a war-injured individual's physical, mental and emotional abilities, limitations, and tolerance in order to identify an optimal outcome for the individual. It evaluates such factors as the soldier's vocational strengths and weaknesses, which in turn could be found in the areas of personality, aptitude, interest, work habits, physical tolerance, and dexterity.[15] Assessment is also prognostic because it attempts to determine whether someone will be able to return to duty and what kind of productive activity the individual will be able to do. An added evaluation goal is to identify those services needed to overcome the functional disabilities that are barriers to successful performance. The specific process of vocational assessment, therefore, is mainly one of diagnosis and prediction that can be integrated into the counseling process and the continued interaction between the hospital healthcare providers and the injured person.

Before varied approaches are used for the vocational assessment of a particular injured soldier, several factors must be considered: the nature of physical and emotional limitations, medication effects, educational experiences, physical tolerance, and the validity and reliability of the specific measures to be used during assessment.

Nature of physical and emotional limitations. These limitations can include communication difficulties, such as visual and hearing impairments, and motor and orthopedic problems. These possible limitations generate for the evaluator such questions as

- What is the extent of impaired manual ability?
- Because of physical limitations, does the manner of recording answers have to be changed? For example, a client may have no use of either hand or, even with use of one hand, may have great difficulty in correctly marking the answer space. Another person may be needed to record the answers to a specially designed answer sheet.
- Do persons with visual impairment have enough vision to handle large objects, locate test pieces in a work space, or follow the hand movements of the evaluator?
- Are assessment measures selected that are appropriate to the individual's level of understanding, particularly his or her reading ability?
- Because someone with an injury that causes serious emotional and cognitive problems will usually have a short attention span, are evaluation measures selected that require shorter tasks?

Medication effects. Specific medicines can hamper the soldier's sustained response to assessment demands, and medications should be checked for their side effects and for their potential impact on performance.

Educational experiences. Most servicemen and servicewomen enter the military with a high school diploma. It may have been many years between formal education or training and injury onset; nonetheless, an exploration of any prior educational successes, such as grades or awards, as well as any specialized training can provide useful data for career planning. The introduction of specific assessment measures, especially in the areas of aptitude and achievement, may cause anxiety which, in turn, could definitely influence vocational assessment outcome.

Physical tolerance. Though discussed earlier with physical limitations, an additional factor is the stamina of an individual as it relates to meeting prescribed time demands of many assessment measures. A person may not have the endurance to complete specific paper-and-pencil tasks. Energy levels and, as already mentioned, tolerance levels, should be explored before assessment commences.

The validity and reliability of the specific measures to be used during assessment. The evaluator, when reading the manual of a particular assessment tool, should explore the normative sample used to develop the measure. If the particular measure is going to be used for predictive, decision-making purposes, the evaluator should consider what the manual states about the reliability and validity coefficients. Many measures traditionally used in vocational assessment have not been normed on the population for which they are being used, for example, ethnic minority populations.[15]

All of these selection considerations imply that to achieve successful vocational assessment, the individual who is conducting the evaluation must work cooperatively with the other healthcare professionals. Information about medications and functional limitations, for example, may have to be provided by a person associated with the individual's care, perhaps in a multidisciplinary team conference.

Because vocational assessment is a comprehensive process that should explore a wide variety of the injured person's characteristics, such as career interests, personality functioning, living behaviors, and physical strengths, there are definite steps in

TABLE 15-2

A COMPREHENSIVE ASSESSMENT APPROACH

Steps	Specific Approach
1. Functional Assessment	Selected measures (Barthel Index - Granger Adaptation; Functional Assessment Inventory)
2. Interview Assessment	Selected questions
3. Family Assessment	Selected questions
4. Interest Assessment	Selected questions and measures (Interest Checklist; SDS; Kuder; CAI; IDEAS)
5. Transferable Skills Assessment	Selected questions and measures in adaptive/self-management, functional, and career content areas (career planning guide)
6. Situational Assessment	Selection of specific in-hospital sites for supervision and assessment feedback

CAI: Career Assessment Inventory; IDEAS: Interest Determination, Exploration, and Assessment System; SDS: Self-Directed Search

the evaluation process that emphasize this multifactorial approach. Career planning should not be based on the assessment of a single attribute such as educational or military service achievement. Other factors, such as family network, adjustment style to disability, or social relationships can be included to learn how an individual is going to adjust to return to duty or to a civilian job placement. This comprehensive approach is identified in Table 15-2 and discussed below.

Functional Assessment

Decisions must be made concerning whether the injured person is physically ready to return to duty, or whether military discharge is more feasible. This assessment includes the attempt to match a person's physical abilities to those required for return to military responsibilities. The functional assessment itself is a series of test activities designed to measure an individual's existing capabilities, limitations, and goals. A dimension often overlooked in the understanding of functional assessment is that this specific evaluation also involves the measurement of behavior. While functional assessment encompasses a very large number of techniques that are impairment oriented, such as the quantitative assessment of muscle strength, it can further include the measurement of behaviors that are involved in essential aspects of everyday life, including personal care (hygiene, grooming, and feeding) and locomotion.[16] These behaviors are collectively known as activities of daily living (ADL), and there are many ADL instruments reported in the literature of medical rehabilitation. Alexander and Fuhrer[16] believe that most available ADL instruments share five characteristics: (1) they measure ability rather than actual functioning; (2) they provide assessments within roughly the mid-range of the behavior spectrum, for example, ranging from the details of buttoning and zipping clothes to eating and ambulating; (3) the instruments are designed to be administered and interpreted by rehabilitation professionals employed in hospital or institutional settings, such as an occupational therapist, psychologist, or rehabilitation counselor; (4) the method of assessment involves ratings made by an observer; and (5) most provide some numerical measure of dependence as an indication of the severity of disability.

Importantly, functional assessment is performed from an environmental perspective; namely, the capabilities or limitations or both are evaluated relevant to the demands placed on the individual's return to military duty or to an appropriate placement in the civilian labor market. Rather than addressing diagnostic labels or individual traits, functional assessment in vocational evaluation examines the specific abilities of the injured person or problems of daily functioning that have direct implications for retraining and eventual return to military duty.

Recommended measures for a functional assessment are (*a*) the *Barthel Index-Granger Adaptation,*[17] an index designed particularly for persons with a physical disability, which includes 15 items that focus on self-care, mobility, and bladder and bowel control; and (*b*) the *Functional Assessment Inventory,* a measure that includes eight scales: cognitive function, motor function, personality and behavior, vocational qualifications, medical condition, vision, hearing, and economic disincentives. Developed by Crewe and Athelstan,[18] it is useful for the beginning formulation of career plans.

Interview Assessment

After a functional assessment has been conducted by a member of the hospital staff, an in-depth, career-oriented interview with the injured person can be undertaken. This session can be the most valuable component of the in-hospital vocational assessment, especially if the individual has severe physical limitations that impede paper-and-pencil testing. The interview can provide insights into the person's understanding of the injury situation, interests, and expectations for a possible satisfying future. Information is frequently communicated during an interview that is often not provided from other assessment devices. Attitudes toward a possible resumption of military duties, for example, may only be learned through the evaluator's observations and insights gained during the interview.

After introductions and communication to the individual that the evaluator is eager to listen and respects the problems the injured person might have, the following areas should be explored:

- Perception of the injury situation and its impact on the development of future plans.
- Expectations the person has for vocational rehabilitation and posthospital discharge: What type of work do you see yourself doing 5 years from now?
- Family history and the injured person's perceived expectations from family members for the individual's resumption of military responsibilities.
- Educational history: subjects liked best and least; any particular achievements.
- Military history: duties liked best and least; any particular achievements.
- Any employment prior to military service: duties liked best and least.
- Leisure-time activities.
- Most difficult problems faced during military service: how were they handled?
- Self-esteem: What do you do well? What do you like about yourself? What do you think you have learned from the injury experience?
- Confidence level: How do you think things can be different for you? What can you do about it? Are you willing to take risks to make changes?

As the individual responds to these questions, the interviewer should observe the person's mood and affect, association of ideas, any shifts in conversations, and recurrent references. All of this information may provide indicators of the injured person's energy, his true feelings about the injury situation, motivation, and expectations for future military service involvement. Apart from the observations, however, answers to the questions identified above provide knowledge about the individual's emotional reactions to the injury, capacity, and confidence to face the problems associated with rehabilitation, and career interests.

The interview experience should assist the injured person to gain an understanding of career options and to appreciate his strengths for handling the injury situation. During this session, the interviewer does not simply ask questions and record information, he also provides verbal support. A skillful interviewer gives frequent "pats on the back" or reinforcement, never openly giving the appearance of cross-examining. Spontaneous comments by the interviewer further help to create a favorable climate for conversation.

Family Assessment

Soon after conducting the initial interview with the injured service person, an attempt should be made to have a brief visit with the person's available family. Family members are often the missing pieces for appropriate adjustment to a sudden injury. Yet, the family can be an important resource for rehabilitation efforts, or represent a major obstacle to their successful achievement. If a spouse or parent of a military service member injured in battle is strongly convinced that the individual should not return to duty, but return to duty is medically feasible, then the injured person is faced with tremendous pressure when making a career decision. Also, as illustrated in the case of Specialist Smith presented earlier in the chapter, a family member can engage in behaviors that actually may be deterrents to vocational rehabilitation. Often the family is not available, but when it is, the following areas can be explored during a family visit in the hospital:

- What information does the family have about the injury condition?
- What are the feelings and expectations of family members about the war injury and the career options for the family member?
- Does the family have any specific needs re-

lated to the war injury of the injured family member?
- What will the family be like if the injured person is discharged from military service, and do family members perceive any specific problems resulting from the injured family member leaving the armed forces?

Because of the briefness of the healthcare provider's opportunity to meet with families, assessment information needs to be obtained at only one meeting. To facilitate the family's verbal expression of important information, it is essential to exercise such basic communication skills as (*a*) attentiveness, (*b*) a nonjudgmental attitude, (*c*) using understandable words when talking with family members, and (*d*) phrasing interpretation tentatively to elicit genuine feedback from family members. The interviewer should not assume anything and should ask only what he believes the family members can answer so that the interviewee feels competent and productive.[15] In other words, the interviewer must create a setting during a family meeting in which people can, perhaps for the first time, risk sharing their emotions and seek information about their concerns. Also, family needs and expectations may change as the injured individual continues in the rehabilitation process. The assessment areas identified above can provide guidelines to help the family be useful partners to the helping professional and to the service member.

Interest Assessment

If there is a possibility that the injured person will not return to his former duties, then the next step in the vocational assessment process is exploration of the individual's interests. This can be conducted using both information and formal approaches, with the latter generally utilized after hospital discharge when the person receives direct assistance from those responsible for career planning. Yet, if an interest assessment is begun while the person is an in-hospital patient, evaluation feedback can be a source of remotivation as well as an initial development of career options.

Various approaches for measuring interests include self-estimation, interviews, checklists, questionnaires, and tests (known as inventories). The choice of assessment approach should be based on the kind of information the particular approach provides, and not simply on availability, low cost, or time required for administration. Prior to beginning interest assessment, moreover, the helping professional should recall any career interest-related information that was disclosed during the injured person's initial interview.

Brief interest exploration approaches should usually be employed in a hospital setting because they require less time than more formal measures and can be scored quickly by the person recuperating from injuries or by the professional helper. Examples of these measures are the *Interest Check List*,[19] the *Vocational Interest, Experience, and Skill Assessment*,[20] and the *Gordon Occupational Checklist*.[21] Though limited in their scope of possible interest areas, these instruments can provide the individual with an early understanding of possible career interest directions. If, however, the service member has the physical and mental capabilities to respond to more detailed inventories, then such interest tests as the *Self-Directed Search*,[22] the *Strong Interest Inventory* (SII),[23] the *Kuder Occupational Interest Survey (Form DD)*,[24] and the *Career Assessment Inventory-Enhanced Version* (*CAI*),[25] could also be used. These tools give varied career options and should be more useful for planning. It should be noted that the SII, Kuder DD, and CAI must be computer scored.

There is still much work to be done for designing more effective interest-assessment instruments for those undergoing in-hospital rehabilitation treatment. But even apart from the difficulties presented by existing interest inventories, the interview can still be employed effectively to solicit information about a person's interests. An approach that could be quite useful in a hospital setting, employing an interview structure, has been devised by Friel and Carkhuff.[26] Their approach involves understanding the client's total functioning in physical, emotional, and intellectual areas, and encourages the person's participation in interest exploration and rehabilitation programming. Most of the steps are designed to elicit the individual's thoughts and feelings about past and future career activities. The six steps of the Friel and Carkhuff approach are presented here formatted for application in a hospital setting.

Step 1. Assist the individual in exploring interests by asking such questions as, From the duties that you have had, what did you particularly like or dislike? In those duties, what do you feel you did especially well? From the people whom you know in your life, what jobs do they have that are of particular interest to you? When you watch television and see people doing various jobs, what jobs do they have that are of particular interest to you? (It is important for the interviewer to understand the reasons behind an identified interest. Is it because of some external pressure, for example, what military superior, family members, or friends told the person he or she would like?)

Step 2. Assist the individual in exploring his values by asking such questions as, When you were performing your military duties, what do you feel was important to you? What is the reason that it was important to you? Was it important to work with your hands, for example, or to have close supervision; or to have the prestige associated with military service; or to know it was something you could do well?

Step 3. Categorize the information that has been generated about the individual's values. Friel and Carkhuff[26] suggest organizing these values into the physical, emotional/interpersonal, and intellectual areas. For example:

- Physical: Dressing well in a military uniform, being outdoors, and performing physical activities.
- Emotional/interpersonal: Having security with the military: having your buddies close by when you were performing your duties.
- Intellectual: Enjoying the opportunity to make decisions.

Step 4. Further categorize the information into:

- People occupations: Includes the areas of service, education, business (salesperson), and providing goods and services.
- Things occupations: Includes technology (providing mechanical services, for example, mechanic or electronic technician), outdoors, and science.
- Data occupations: Includes the areas of data entry and analysis, record keeping, and computation.

Step 5. Help the individual identify which of the people, things, and data interest categories fits his values. For example, if the person indicates that job security is the most important career value, an interest area previously identified within Step 4 and that is in harmony with offering job security, could be discussed.

Step 6. Identify the educational and occupational requirements demanded of particular career areas that are congruent with the individual's values.

Interest assessment is an integral part of vocational assessment and suggests which alternative courses of action are potentially satisfying for the rehabilitant. Within the hospital much information about these interests can be gained from an effective use of the interview.

Transferable Skills Assessment

With many battle injuries, it may take some time for a person's physical condition to stabilize and thus permit any predictive career assessment. But as the individual is drawing close to hospital discharge and a determination is being made about career directions, the person should participate in a transferable skills assessment. Even if the person is going to remain in the military service but have different responsibilities, this specific assessment can be especially valuable. If, however, the individual is going to be discharged from the military service because of his injuries, then an evaluation of transferable skills is all the more necessary.

Transferable skills can be grouped into three categories: adaptive/self-management skills; functional skills, and career content skills. Examples of each area are shown below.

Adaptive/self-**m*****anagement skills.*** Personal management in relation to authority, punctuality, dress, care of property, impulse control, dependability, initiative, and resourcefulness.

Functional skills. These comprise physical, intellectual, aptitude, and achievement skills the soldier has either as innate talent or acquired by specific educational, vocation, or avocational special training. Such skills could include effectiveness in dealing with many kinds of people, for example, being "a natural salesperson," or possessing artistic talent.

Career content skills. These skills focus on those abilities related to performing a job in a particular field, profession, or occupation, and usually are acquired through technical or specialized training prior to injury onset, whether in the military or a school before service entry.

An exploration of these skills with the individual is an identification of the specific ways that intellectual and physical abilities can be used to perform specific career tasks. There are different inventories available that simply require the person to check those skills which he believes he has and can utilize for other military service or career goals. Two inventories are particularly useful: *Operation Job Match*[27] and *Career Planning Guide Book 3*.[28]

With the use of inventories, questions can be developed for the interview that will provide information on the individual's perceived skills. Such questions are:

- What projects have you accomplished? What skills did they require?
- Have your military accomplishments made you aware of special abilities? What are they?
- What do other people recognize as your skills?
- While in the military, have you taken on especially difficult tasks, ones that others didn't want to do? Which ones?

Implied in these questions is that all people have some form of excellence within them that will be expressed in experiences they feel to be achievements or successes. Examination of these many experiences will reveal a pattern of skills that are used repeatedly. For a more comprehensive understanding of an individual's skills, usually both verbalized questions and a written inventory should be used together. Frequently, individuals will trivialize their experiences and accomplishments during the interview or take them for granted, but a checklist or inventory can act as a stimulus for information disclosure that indicates skill patterns and priorities. When available, service records of assignments and performance ratings should also be used as a source of information.

Situational Assessment

When an opportunity is available within the hospital setting to place the recuperating person into a work location where one performs designated tasks, the experience can provide valuable information about career-related behaviors. Situational assessment is essentially the observation of people in work situations, and is one of the most commonly used techniques for vocational evaluation.[15] This assessment emphasizes work behaviors that are observed over a period of time in an environment that is as closely realistic as possible to actual occupational demands. This type of evaluation permits the individual to learn again the role of the worker, allows the evaluator to assess many more work behaviors that can be explored with standardized vocational testing, and minimizes the typical test-situation anxiety.

For situational assessment to be effective, an appropriate, in-hospital site should be utilized, adequate supervision provided, and a means used to gather information that, in turn, can be translated into rehabilitation planning. Because the observational approach is the basis of situational assessment, these observations must be carefully planned and scheduled, and well-designed rating and observation forms should be used. This demands that the evaluator understands the possibilities for on-site assessment within the hospital and identifies supervisors who can reliably complete an observation form. If these requirements are met, the information gained from situational assessment is added knowledge for the person recuperating from injuries to use in developing realistic career options.

The components of an effective vocational assessment program within the hospital are functional ability, interview, family, interest, transferable skills, and situational. The approaches representing the understanding of functional abilities, using an interview, and obtaining family information can be implemented as early as possible after admission to the hospital, as can the process of obtaining the person's prior service record. Healthcare related personnel available in the hospital are usually capable of conducting any of the six suggested assessment areas. Rehabilitation counselors, occupational therapists, and counseling psychologists have all received training in assessment. With additional preparation in specific vocational assessment methods, especially in the use of transferable skills and situational observation techniques, they can perform an assessment that generates useful information.

Particular medical problems, such as head and spinal cord injuries, as well as injuries resulting in amputations, will present unique problems for an evaluator. The recovery period is considerably longer than that demanded by other war injuries, and the associated severe pain of these wounds can have a decided impact on career planning. Because of the physical limitations accruing from these injuries, the interview will most frequently be the method of choice for vocational assessment. The timing for any career assessment is another consideration. Reliable evaluation results will not be gained until these conditions have medically stabilized, and it is perceived by medical personnel that there will not be a drastic change in the individual's physical condition. Yet, traumatic brain injuries (TBIs) and their residual effects may still suggest an uncertain physical, intellectual, and emotional vocational adjustment for the individual. Interests and transferable skills can change during the course of recuperation, and perhaps the most feasible in-hospital assessment is a situational evaluation just before hospital discharge, followed by a more realistic vocational assessment during outpatient status. Frequently, the evaluations on persons with TBI cannot be conducted until 18 to 24 months after injury onset.

Another specific injury that offers particular assessment demands is a wound resulting in low back pain. This injury can bring persistent pain, even after intensive, medical treatment has ceased; and though the physical source of such pain may often not be identified, it is still real to the individual. All six assessment areas suggested in this chapter are applicable for the evaluation of the person with low back pain, with perhaps an added emphasis on functional and family assessment considerations. Persistent pain has a strong influence on personality and behavior, and functional assessment may identify some of the negative manifested behaviors. Additional assessment measures, however, that explore personality dynamics may have to be used. For this purpose the Minnesota Multiphasic Personality Inventory (MMPI-2)[29] is especially valuable. Also, because the individual's pain can additionally impact the person's family and family members may perceive that they will have to live with someone who is attempting to cope with a troublesome injury, family expectations and possible family disincentives should be explored. If family members believe that a return to duty or a particular career option may aggravate an existing injury, then they may thwart any career planning efforts.[30]

Following counseling and vocational assessment, plans are usually developed for an individual. In harmony with the importance of early intervention, career planning should be done before hospital discharge. Careful attention should be given to the development of these plans. Detailed career planning is a means of communicating that a career is feasible both to the individual recuperating from the war injury and to the one who will follow up on the person's rehabilitation after discharge.

The individual's career plan is formulated after evaluation results are shared and feedback received on the person's perception and meaning of the assessment results. The evaluator gathers all the information acquired from each of the six areas, or those areas that are utilized; assembles a career profile of the individual's strengths, needs, interests, and possible career-related obstacles; and explains this profile to the individual. Career goals are then identified, and a plan to reach these goals is developed. The guidelines for a career plan are shown in the Figure 15-1.

CAREER PLAN GUIDELINE

1. Major goal: ______________________________

2. Subgoals (steps needed to achieve major goal): ______________________________

    A. (Subgoal) ______________________________

        Date of Implementation: ______________________________

        Date of Completion: ______________________________

        Resource: ______________________________

        Monitor: ______________________________

    B. (Subgoal) ______________________________

        Date of Implementation: ______________________________

        Date of Completion: ______________________________

        Resource: ______________________________

        Monitor: ______________________________

    C. (Subgoal) ______________________________

        Date of Implementation: ______________________________

        Date of Completion: ______________________________

        Resource: ______________________________

        Monitor: ______________________________

Fig. 15-1. An example of career plan guidelines.

POSTHOSPITAL PHASE

Earlier in this chapter, the vocational rehabilitation process for war-injured service personnel was conceptualized as taking place in two phases. However, it is difficult to view this process as really breaking down into two distinct periods. It has been emphasized that the vocational rehabilitation process should begin as early as possible after injury onset; and this process will continue after discharge from the hospital, though different issues should receive attention. A few of these issues are the same whether the individual is returning to military duty or is entering civilian life and preparing for civilian career placement. To be noted is that the Department of Veterans Affairs (VA) is aware of the importance of a continuity of vocational services, and has developed the Transition Assistance Program to respond to this need. A transition assistant specialist is now located in many service-related hospitals, and meets with the recuperating individual to begin planning options for after–military-service discharge. The program consists of vocational assessment and job search assistance. Within each area the individual receives information about vocational capabilities, with an emphasis on interests, transferable skills, and work-related values. More detailed information about how to conduct a job search, including the identification of potentially helpful community resources is also provided. Initial contact with military service personnel is usually made as the time for hospital discharge approaches.

There are four issues that frequently need attention after the individual leaves the hospital: (1) posttraumatic stress disorder, (2) work hardening, (3) return-to-duty transition, and (4) the coordination of necessary services. Langley et al[31] explain that many persons might be viewed as coping effectively with their posttraumatic stress simply because they have few if any reported vocational or job-related difficulties. In reality, duty or job performance may only serve as a defense against the nightmares, intrusive thoughts, and other symptoms of posttraumatic stress disorder. Over time, this defense may become less effective as the pressures arising from the individual's illness continue. Therefore, the need for professional assistance emerges.[31] Unfortunately, this illness may be exacerbated by a wide variety of causes, such as societal/community/family disapproval of the specific war or conflict, disappointment in a career, or guilt or shame over circumstances related to the injury. Whatever the cause or the intensity of symptoms, professional attention is needed that addresses the unique problems and conveys both understanding of the individual's pain and defense mechanisms and useful insights into problem resolution. The process of assessment is critical in posttraumatic stress disorder. The initial assessment interview conducted after hospital discharge, when the soldier's symptoms become apparent, is the first setting in which the real nature of the trauma can be confided and the process of unburdening distress can take place. The presence of another person can play an important role in evoking hope and trust; key factors for the soldier when beginning therapy for this life predicament.[32]

Work hardening is another issue for many individuals who may be completing the recovery process, but still are not ready for the full resumption of military duties or civilian career placement. A relatively recent service in the vocational rehabilitation process, work hardening emphasizes (*a*) work simulation as a primary component, which includes exercise, aerobic conditioning, and education, and (*b*) a multidisciplinary approach (physical therapy, occupational therapy, vocational counseling, and psychology). When work hardening programs are available, it is more conducive to a successful return to duty, or to an appropriate civilian job placement, if the program is begun as early in the recovery period as possible, before the individual is discharged from the hospital. There are several programs available in large, urban areas that can respond to the needs of the veteran.

A third issue is specific to the soldier returning to military duty and comprises the nature of the transition from in-hospital treatment to an outpatient status and perhaps light duty demands. Returning to duty may be a time of personal crisis for many soldiers. A soldier may still harbor guilt and anxiety over the circumstances of an injury, and his expectations for a successful military career may be changing. These expectations may be reinforced by a spouse or other family members who are disappointed in the resumption of a military career. Consequently, at the time of hospital discharge, the soldier should be given the opportunity to explore and to disclose any feelings about returning to duty, and arrangements should be made, when necessary, for support in the individual's unit. Support can take the forms of listening to concerns, reinforcing feedback related to duties performed, and indicating that professional help is available to the soldier for emerging difficulties. To encourage this support, communication is often necessary between the medical staff and the person's military unit. Also, follow-up should be conducted to assess whether the soldier is following a posthospital, treatment regimen. When the details of the regimen were communicated at the time of hospital discharge, the soldier's anxiety about returning to duty may have inhibited the understanding of important information.[33]

The fourth issue thematic to both the individual returning to duty, or to the person leaving the military, is the coordination of necessary services. Vocational assessment and counseling will often reveal many concerns that will not be resolved during in-hospital recuperation from an injury. Several emotional concerns, such as continued feelings of anxiety, fear, and loss; family matters; and assistance for career planning or job search all demand coordination so that services can be delivered in a timely, organized manner. This coordination should be arranged by a designated person at the time of hospital discharge, or very soon thereafter.

Once it has been definitely decided, however, that the individual is not going to return to any military duties but will be discharged from the service, certain steps leading eventually to an appropriate career placement should be taken. These steps begin around the time of the individual's departure from the hospital and continue until after he leaves active military service. One of these steps includes contacting a Veterans Benefit Counselor at the nearest VA Regional Office. The veteran is eligible for the VA vocational rehabilitation program, which provides services and assistance necessary for service-connected disabled veterans to achieve maximum independence in daily living and to obtain and maintain suitable employment. Vocational rehabilitation can include college, technical school, or on-the-job training. Rehabilitation services include assessment, counseling, training, subsistence allowance, and employment assistance. The VA will assist in job placement; and while a veteran is enrolled in a vocational rehabilitation program, the VA pays the cost of tuition, fees, books, supplies, and equipment. The VA may also pay for special supportive services, such as medical and dental care; prosthetic devices; and lipreading, signing, and training for those who are hearing impaired.

A veteran with other than a dishonorable military discharge is entitled to vocational rehabilitation benefits if the VA determines that the veteran needs vocational rehabilitation to overcome a medical problem that affects his ability to prepare for, obtain, or retain employment consistent with abilities, aptitudes, and interests. The veteran's service-connected disability must contribute to this employment handicap.

Eligible veterans who are disabled may receive rehabilitation services for a designated period of time. Generally, the veteran must complete a rehabilitation program within 12 years from the date the VA notifies him of entitlement to compensation benefits.

CONCLUSION

This chapter has explained a proposed process of vocational rehabilitation for the person injured during a military conflict, who is recuperating in a military hospital. The emphasis during this process is on attention to the emotional effects of the casualty, counseling, and vocational assessment. Important for the success of the individual's return to military duties, or to civilian placement following service discharge, is early, in-hospital intervention. Resources have been made available by the military services to assist the person in making an appropriate life adjustment. It is suggested that the existing career resources be implemented as soon as possible after injury onset. In some locations, some of these resources for career development may have to be modified or created, but the provision of timely, comprehensive intervention is necessary to fulfill the legal and moral obligation to those who have served.

Equally important to successful vocational rehabilitation outcome is the soldier's positive motivation and emotional investment in the process. Here again, the sooner after injury work begins with the soldier toward these goals, the easier and more effective their attainment will be.

Finally, the earlier that the rehabilitant's family, social support system, and eventual career placement setting (in or out of the military) can be brought into the planning process, the easier the posthospital discharge transition will be. Our society has gone a long way toward defining the rights of all citizens with disabilities, and those with service-connected disabilities and their advocates should become aware of and ensure compliance with these rights.

In final analysis, successful vocational rehabilitation requires a motivated client and a facilitative environment. These are best achieved by early intervention and carefully planned coordination of services.

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Chapter 16

THE US ARMY PHYSICAL DISABILITY SYSTEM

CHARLES A. PECK, Jr, M.D.*

INTRODUCTION

HISTORY
    Early Development
    20th Century

THE US ARMY DISABILITY SYSTEM
    Medical Evaluation Board
    MEB Narrative Summary
    Review of MEB NARSUM
    The Physical Evaluation Board
    The PEB Process
    Review by the USAPDA
    Final Outcome of the Review by the USAPDA
    Statistics Illustrating the Function of the Army Physical Disability System

THE US NAVY AND AIR FORCE DISABILITY SYSTEMS
    US Navy
    US Air Force
    Comments

CONCLUSION

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INTRODUCTION

When rehabilitation of a soldier is not completely successful after injury or illness, it may be necessary to consider separation or retirement for reasons of permanent medical disability. Historical data indicate that 10% to 15% of surviving combat casualties undergo disability separation.[1-4] In addition, separation or retirement due to permanent medical disability may be necessary for soldiers who are injured by the rigors of training in preparation for combat, or as the result of accidents or illnesses.

The disabled soldier must be remembered. He deserves and must receive acknowledgment of hardships endured and perils he has survived. His spirit is never *hors de combat* and to cast him aside will ensure that few kindred spirits will follow him into a life dedicated to the defense of his nation. Since the beginning of military history, many rulers and governments have understood this principle. Historically, they either established pension systems or demanded that those who were responsible for furnishing troops also provided a system of fair, equitable compensation.[5] The army physical disability system has been established to compensate soldiers who have injuries or disease that have terminated their military careers. The purpose of this chapter is to describe, first, how the United States Army disability program is organized and performs its mandated functions, and second, some of its more recent accomplishments.

HISTORY

Early Development

Many ancient civilizations had compensation systems. The law of Ur-Nammu, King of Ur in Sumer circa 2050 BC, was believed to be the first of such laws.[6] This law was closely followed by the code of Hammurabi.[7] In 1592, the British Parliament provided yearly pensions limited to £10 for privates and £20 for lieutenants. Pensions were for those who "have adventured their lives and lost their limbs or disabled their bodies, to the end that they may reap the good fruit of their deserving, and others may be encouraged to perform like endeavors."[8] This is also an early example of soft-sell recruiting. Some 90 years later, England established a standing army and instituted a system of soldiers' homes to care for superannuated enlisted men. Officers mustered out of the service were to be carried at half pay status and remain liable for service in the event of war. The British system provided the foundation for the military retirement system of the United States of America.[5]

The first national pension law in the United States was enacted by the Continental Congress on 26 August 1776,[9] just 7 weeks after the Declaration of Independence had been signed. It provided half pay for life for all ranks of disabled veterans. Because many officers were not able to perform their duties due to old age and chronic illness, a law was enacted in 1855 that was initially applicable only to navy officers[10] and which permitted retirement after 40 years of service upon application to the President. A similar law, applicable to the army, was enacted in 1861.[11] This same law allowed those officers who became physically incapable of performing duty to be placed on the Reserved List. Retired pay was 75% of active duty pay. In 1862, a law similar to the current law was enacted[12]; it applied to both Army and Navy and provided pensions according to rank for all who were totally disabled. Partially disabled soldiers received proportionate amounts; widows; children under 16 years of age; dependent mothers; and orphaned, dependent sisters could receive total disability pension.

20th Century

Over the years, the pension laws were broadened to extend coverages, but the next major advance in military compensation would have to await the flood of disabled soldiers and sailors caused by World War I and World War II. In 1928, after World War I, a law was enacted that applied to all officers of World War I.[13] Soldiers who had more than 30% disability were placed on the Emergency Officers Retired List at 75% of the active duty pay to which they were entitled. Those with less than 30% disability were also placed on an Emergency Officer Retired List, but without pay. All were entitled to other benefits and privileges of retired officers.

The Veterans Bureau was established in 1921.[14] Its task was to administer benefits for non-Regular Army officers. In 1939, this law, as it applied to non-Regular Army officers, changed. If non-Regular Army officers were called to active duty in excess of 30 days and suffered disability or death in the line of duty, they were entitled to the same benefits as Regular Army officers.[15]

The law, however, was not applicable to enlisted soldiers. This oversight was corrected in 1941 by legislation affecting Regular Army enlisted soldiers with more than 20 years of service and with permanent disability.[16] They became eligible for placement on the Disability Retired List and were to receive 75% of the average of their last 6-month's pay just prior to becoming disabled.

When the United States entered World War II, no disability retirement law was in place to cover the more than 10 million soldiers who would participate in this great war. The Career Compensation Act of 1949,[17] specifically Title IV, provided a law uniformly applicable to the three military services. This was later codified in Chapter 61, Title 10, of the United States Code (U.S.C.). It established rules of entitlement for members of the three services who were unfit for duty because of a disability incurred while on duty for more than 30 days, and a separate set of rules for those called to active duty for less than 30 days. This law provided benefits for Regular and Reserve officers and enlisted personnel, if the disability was due to injuries in the line of duty (LOD). The Act also provided severance pay to soldiers whose disabilities were not of sufficient degree to qualify them for retirement but did interrupt their careers.

A new addition provided to the 1949 Act[18] allowed placement of the soldiers on the Temporary Disability Retired List (TDRL). In the strictest application of this provision only soldiers who had medical conditions that might resolve and thus permit a return to active duty were eligible to be placed on the TDRL. In 1951, the law was interpreted by the US Department of Defense (DoD) to include all soldiers whose medical conditions were unstable regardless of whether they might return to active duty.[19] Stability was defined as no change in rating over a 5-year period. There is a 5-year limit for time on the TDRL. After 5 years, or less if the soldier's condition appears to have stabilized, final disposition is to be made.

Under the Compensation Act,[18] the secretary of each service branch was responsible for implementing the disability compensation process. Prior to this, the Veterans Administration had made the disposition in cases of soldiers with disabling conditions. Initially, the army process was implemented at the medical treatment facilities (MTFs). Alleged inequities in the system led to a review by the Secretary of the Army in 1965 and resulted in the establishment of the United States Army Physical Disability Agency (USAPDA) to implement the US Army's compensation program. Implementation was accomplished in accordance with Title 10, United States Code, Sections 61, 133(b), 1201, 1203, 1210, 1216(d) and 3010[18]; as well as by the Department of Defense (DoD) Directive 1332.18[19]; and Army Regulation (AR) 635-40.[20(ch3,¶2b(1))] "Separation from the Military Service by Reason of Physical Disability," a revision of DoD Directive 1332.18[21] and additions to Army Regulation 635-40,[22] have provided additional policy guidance. The rationale behind the laws establishing USAPDA are to be found in the following quotations:

> The mission of the army physical disability system is to 'provide a full and fair hearing to determine soldiers physical fitness for continued military service, determine level and type of compensation, and take action to separate or retire soldiers when a career is interrupted by reason of physical disability.'[23]

> Disability compensation is not an entitlement acquired by reason of service-incurred illness or injury.[20,22]

> The mere presence of an impairment does not, of itself, justify a finding of unfitness because of physical disability.[20,22]

THE ARMY DISABILITY SYSTEM

The army physical disability system depends on the function of two distinct entities: (*a*) the Medical Evaluation Board (MEB), which is transient and is called into being when and where it is needed, and (*b*) the Physical Evaluation Board (PEB), which has a permanent existence. The orientation and the organizations of the MEB and the PEB together with the controlling hierarchy of the USAPDA differ. Although both boards evaluate a soldier's medical impairment, the MEB uses strictly medical standards and the PEB uses military performance standards (Figure 16-1). While the MEBs can be constituted at any MTF, the USAPDA began with six regional PEBs and a Central Army Physical Review Council composed of four physicians; two lawyers; two personnel management officers (PMOs); a director and a deputy director, with an administrative staff; and a Physical Disability Branch (PDB). Currently, due to the alteration in force structure (downsizing) of the army, the USAPDA has three

Fig. 16-1. The primary components of the Army Physical Disability System, the Medical Evaluation Board (MEB) and the Physical Evaluation Board (PEB), use different criteria to evaluate the potentially disabled soldier. The MEB uses medical standards, while the PEB uses the soldier's performance of his duties to determine fitness.

Fig. 16-2. The organizational aspects of the Army Physical Disability System. Note that the Medical Evaluation Boards (MEBs) report to the Physical Evaluation Boards (PEBs), with the Physical Evaluation Board Liaison Officer playing the essential role as intermediary. The PEBs, but not the MEBs, are components of the US Army Physical Disability Agency (USAPDA), which in turn, is part of Personnel Command (PERSCOM). The MEBs are part of the Medical Command (MEDCOM). At the highest level in the army, the Army Physical Disability Appeals Board (APDAB) exists to advise the Secretary of the Army on the appropriate disposition for the small fraction of cases that are not resolved at the USAPDA level.

AMC: Army Medical Center
MTF: medical treatment facility
PEBLO: Physical Evaluation Board Liaison Officer

MTF: military treatment facility
PEB: Physical Evaluation Board

Fig. 16-3. There are four ways a soldier can enter the Army Physical Disability System: (1) physician, (2) Military Medical Retention Board (MMRB), (3) commanding officer, and (4) higher command. Once in the system, procedures are structured to give the soldier the correct evaluations.

regional PEBs and a PDB. The USAPDA is presently located in Building 7 at Walter Reed Army Medical Center, Washington, DC. The PEBs are located at Walter Reed Army Medical Center, Washington, DC; Fort Sam Houston, San Antonio, Texas; and Fort Lewis, Washington. With continued restructuring of the army, this configuration is likely to change, but the basic relationships remain: a multitude of MEBs are forwarded to a limited number of PEBs (Figure 16-2). Finally, USAPDA with its PEBs, but not the MEBs, is part of the Total Army Personnel Command (PERSCOM).

Both of these boards are proponents for the soldier. It should be noted that it is an informal custom to refer to not only the physical entities of the MEB and the PEB, but to their deliberations and to the products of their deliberations, as "boards." Thus, the physical entity of, say, the MEB is the site of a MEB, which produces a MEB.

There are four ways a soldier can enter the army physical disability system (Figure 16-3): (1) The physician caring for the soldier may refer a soldier after determining that the soldier does not meet medical retention standards. Retention standards are those guidelines put forth in AR 40-501.[24(ch3)] These are medical conditions that may preclude retention in the army. (2) Soldiers who have a Permanent 3 or Permanent 4 profile are required to appear before the Military Occupational Specialty-Military Medical Retention Board (MOS/MMRB), which may recommend that the soldier enter the army physical disability system. (3) A commanding officer may request through the medical commander, that a soldier undergo a fitness-for-duty evaluation. (4) Entry is directed by higher command (eg, the Secretary of the Army or his designee requests a fitness-for-duty evaluation of the soldier). Approximately 85% to 90% of the MEBs are physician directed and 8% to10% are MMRB directed. Soldiers may not direct that a MEB be convened on their behalf.

Medical Evaluation Board

Soldiers, in the performance of their duties, may incur injuries with residuals that prevent them from adequately meeting retention standards.[24(ch3)] An injury may be an acute injury, chronic residuals of injury or illness, or chronic and recurring illness or injury that is repeatedly exacerbated by the nature of the soldier's duties. Diseases may also be acute and debilitating (eg, a myocardial infarction) or chronic and recurring (eg, asthma, arthritis, or diabetes). The soldier is treated at a military hospital for the acute episode, after which a period of convalescent leave may be granted. During this convalescent period the soldier is still considered a patient of the hospital and must return to the hospital on a periodic basis to be evaluated by a physician. The physician determines if he can return to full duty, partial or restricted duty, or should undergo an MEB. The soldier who can return to duty with restrictions, is given a physical profile. The physical profile is a written communication between the soldier's physician and his commander that explains the soldier's medical condition and how the condition might affect duty performance.[24(ch7)]

Unfortunately, the physician all too often writes the physical profile in such a way that it remains unclear as to exactly what the soldier is or is not allowed to do when he returns to duty. The resultant miscommunication leads to inappropriate restrictions on the soldier. Conversely, sometimes the profile fails to reach the commander and demands are placed on the soldier that may be detrimental. Currently, these problems are being reassessed. A profile is either temporary, to be reevaluated at a given date, or permanent, to remain with the soldier for the rest of his career unless altered by the MTF profiling officer and an appropriate profile board according to AR 40-501.[24(ch7)]

In some cases, the soldier may not meet retention standards. If this decision is based on the observed outcome of a disease or an injury, an MEB must be convened. Once the physician and the soldier have agreed that an MEB should be conducted, the Physical Evaluation Board Liaison Officer (PEBLO) of the hospital is contacted. The PEBLO will counsel the soldier and the physician on MEB procedures and monitor the process, the product of which is the MEB Narrative Summary (NARSUM) (Figure 16-4).

According to AR 40-400[25(ch6,p60)] and AR 635-40,[22] MEBs can be established at any MTF and are convened to document a service member's medical status, to review possible duty limitations, and to determine whether a soldier meets medical retention standards. They do not determine fitness or unfitness for military service. The MEB is made up of two or more physician members. One of these must be a senior medical officer with detailed knowledge of (*a*) directives pertaining to standards of medical fitness and medical disposition of soldiers, and (*b*) disability separation processing and the Veterans Administration Schedule for Rating Disabilities (VASRD).

Where dental or psychiatric conditions are of major importance, a dental officer and/or a psychiatrist must be a member of that MEB. Where mental competence is in question, the MEB will consist of at least three members, one of whom is a psychiatrist. The completed evaluation, the MEB NARSUM, is referred to the approving authority, which may be the hospital commander, but is most likely to be the Deputy Commander for Clinical Services, who will review the board's findings for completeness, accuracy, and logic. It has been suggested that the MEB NARSUM have a standardized format for easier review. It is the responsibility of the referring physician to prepare the medical documentation that is to be presented to the MEB. A suggested format follows.

DCCS: Deputy Commander for Clinical Services
MEB NARSUM: Medical Evaluation Board Narrative Summary
PEBLO: Physical Evaluation Board Liaison Officer

Fig. 16-4. To determine if a soldier meets medical retention standards, the Medical Evaluation Board is made up of appropriate medical personnel.

MEB Narrative Summary

Military History

The physician opens the MEB NARSUM with a statement indicating why the review is being done, that is, it is physician directed, MMRB directed, or appropriate command authority directed. This is followed by information on the administrative status of the soldier (ie, active duty, reserve component, or National Guard), which establishes eligibility for board proceeding. This information and the soldier's pertinent military history will have been supplied to the physician by the PEBLO. Included here should be data concerning the soldier's current status, such as mandatory retirement, selective early retirement, bars to reenlistment, and any pending administrative actions.

If the soldier's medical condition is the result of injury or alleged injury, an LOD investigation report must be included, indicating whether the injury was incurred in the LOD or was due to the soldier's misconduct, willful neglect, or both and not in LOD.

It is imperative that this determination be made early. It in no way implies that the soldier will not receive medical care, but the lack of a LOD determination can delay processing and finalizing of the soldier's case. It is best to prepare the LOD investigation report early while facts are still clear in everyone's mind.

Soldier's Chief Complaint and History of Present Illness

Following the military history, the soldier's chief complaint is recorded in his own words. A detailed history of the present illness elaborating the chief complaint is next. If the illness is chronic, documentation of the chronological sequence is important to ascertain how the medical condition has affected the soldier, how military service has affected the condition, and what the impact has been on duty performance. A soldier who has had a chronic knee problem all his military career but has been able to do his duty up to retirement may be viewed differently than a soldier who has an acute knee injury requiring operative intervention. It is incumbent on the physician to document how a soldier's medical condition affects his duty performance.

Past Medical History

After compiling a concise chronological history of the present illness, an equally concise pertinent past medical history is important. Any illnesses that were present prior to service entry are noted. Of major importance would be any prior hospitalizations and operations. A good source for this information is the Induction Medical Form (Report of Medical History, Standard Form 93) shown in Figure 16-5. The past medical history is vital in many disease processes, especially when the soldier may have a psychiatric illness. The relevant family and social histories are also included in the past medical history.

Current Physical Examination

After the pertinent history, a complete physical examination is recorded. The examination should not be only qualitative as related to the soldier's chief complaint, but should also be quantitative whenever possible. A qualitative finding study is a subjective type of finding, which does not have support other than the physician's observation. A quantitative finding study contains not only the physician's opinion but also factual data (measurements) to support the opinion. Quantitative findings carry a greater weight in the final decision as opposed to qualitative findings. Pertinent consultative evaluations are obtained when necessary. These may be used in the physical examination or as addenda to the MEB NARSUM.

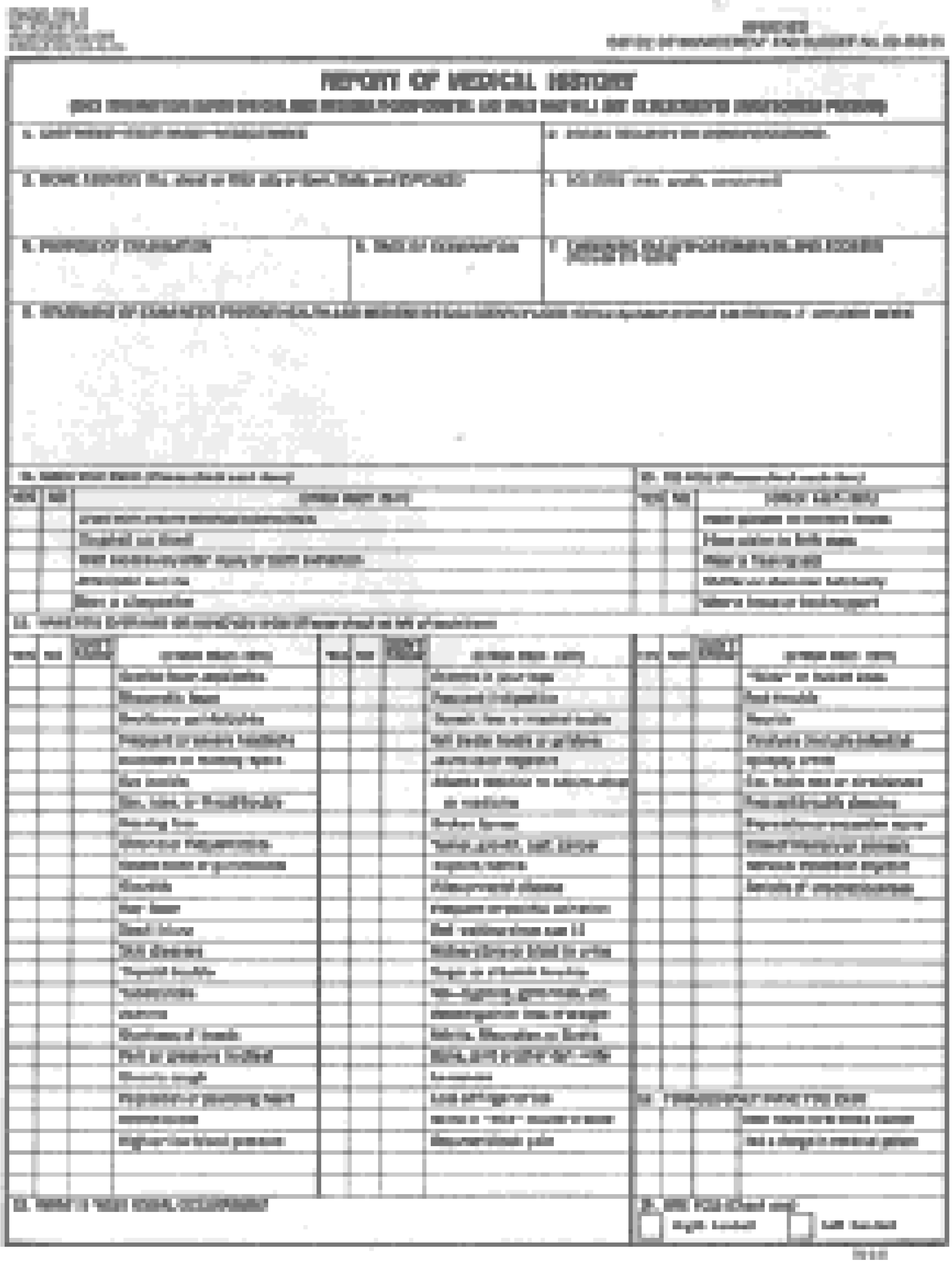
Quantitative evaluations of various systems include: (*a*) pulmonary function studies for respiratory disease or thoracic surgery cases, (*b*) stress tests and angiogram studies in cardiology cases, (*c*) early and late neuropsychiatric testing in head trauma cases, and (*d*) electromyographic and/or nerve conduction studies in cases involving neurology, neurosurgery, and physical medicine. In addition, careful physical examination with supporting radiographs (plain films, computed tomography [CT], magnetic resonance imaging [MRI] studies) and muscle and nerve conduction studies are incorporated into the overall assessment. It is mandatory in orthopedic cases that range-of-motion studies be recorded if joint involvement is a factor. If one of paired extremities is uninjured, this presents an excellent standard for comparison to the injured extremity for range-of-motion and function.

The physician, having obtained a thorough history of all relevant aspects of the patient's military experience and medical complaints, and a physical examination, then records laboratory data to support the diagnoses. In addition to the general studies obtained, special studies such as those mentioned above may be included here.

Soldier's Assignment

The soldier may or may not have been hospitalized for the board process. If he was hospitalized, a chronology of the hospital course should be entered. If he was not hospitalized, he may be assigned back to his parent unit or to the MTF's medical holding unit. All hospitalized, active-duty, uniformed patients within an MTF's geographical region are attached to the medical holding unit (each inpatient MTF must have such a unit[25(ch7,p67)]); this information about the soldier's assignment should be included as part of the MEB NARSUM. The soldier's duties and how well the duties were performed while he was assigned to his parent unit or the MTF medical holding unit are also noted.

When the patient completes the hospital course, the physician records the soldier's current status and prognosis for recovery or partial recovery from the medical condition. A cardiac patient recommended for a trial of duty should have this recom-



REPORT OF MEDICAL HISTORY

Fig. 16-5. The Induction Medical Form, (Report of Medical History, Standard Form 93), which is a good source for obtaining information on the soldier's medical problems prior to his entering the army.

Fig 16- 5. *continued*

mendation recorded in the hospital records. If the soldier was placed on convalescent leave, his progress during this leave should be documented.[26(ch5,§3,¶5,6)] AR 600-8-10 defines convalescent leave as a period of time, not chargeable to regular leave, given the soldier to recuperate, or convalesce due to injury or illness. Up to 2 weeks of convalescent leave may be granted by the unit commander. In most instances, however, the duration of leave is recommended by the soldier's physician. The soldier's physician may request up to 30 days of leave. Leave in excess of 30 days must be approved by the hospital commander or his representative. The soldier on convalescent leave is still considered a patient of that MTF.

Medical Diagnoses and Conclusions

All medical diagnoses should be listed, beginning with the most critical. If possible these should be correlated with the VASRD as closely as possible. If the disease is not present in the VASRD, current medical diagnoses should be used. The physician then records conclusions as an ending to the MEB NARSUM. Conclusions should state whether the soldier meets retention standards according to AR 40-501 chapter 3 and cite the appropriate paragraph. Using paragraph 41e does not provide adequate information for the PEB and is used only when the physician does not have an accepted medical condition to cite. AR 40-501 chapter 3 paragraph 41e refers to "miscellaneous conditions and defects: (1) conditions that result in interference with satisfactory performance of duty as substantiated by the individual's commander or supervisor; (2) the individual's health or well being would be compromised if he were to remain on active duty; (3) in view of the soldier's medical condition, his retention in the military service would prejudice the best interest of the Government."

Review of MEB NARSUM

The attending physician, having competed and reviewed the MEB NARSUM, signs it and meets with a senior medical officer for review and signature. As mentioned previously, if the board has been convened to review psychiatric impairment or mental competency, an additional physician—a psychiatrist—is required at this meeting to review, agree, and sign the MEB NARSUM prior to forwarding it to the approving authority. If dental impairments exist which preclude retention on active duty, a dental officer must sign the MEB NARSUM. Once the approving authority has signed the MEB NARSUM, the soldier is then counseled by the PEBLO regarding the board's results. The soldier has the options of agreeing with the board's results or disagreeing. If he does not agree, the soldier must submit within 3 working days the reason for his disagreement. This written statement is reviewed by the approving authority and the MEB NARSUM is either (*a*) returned to the physician for compliance with the soldier's request, (*b*) forwarded to the PEB with the disagreement statement noted by the approving authority, or (*c*) forwarded to the commander for review. If the commander is the approving authority, the MEB NARSUM may be forwarded to the next higher command for resolution.

The Physical Evaluation Board

The soldier, thus having completed the first major step in the army physical disability system, will have been (*a*) found to meet medical retention standards and returned to duty with profile as warranted, or (*b*) forwarded to the PEB through the PEBLO, because retention standards were not met. Soldiers who do not meet retention standards are not necessarily unfit.[22(ch3-1)] A fit or unfit determination is made by the PEB. It is important for the medical officer to understand that medical impairment is not necessarily synonymous with, or the cause of, physical disability. No two soldiers are identical and no two cases presented to the army physical disability system are the same. In cases of grave medical conditions, objective medical evidence is sought and is weighted more strongly in the evaluation done by the PEB. In cases of chronic, long-term medical conditions, more weight is given to subjective complaints, performance, and administrative data when determining whether a soldier is fit or unfit (Exhibit 16-1).

The PEB differs from the MEB in many aspects but primarily in the basis of its findings. A MEB determines if a soldier meets medical retention standards according to AR 40-501, chapter 3. The PEB determines whether a soldier is fit or unfit, taking into consideration the medical findings of the MEB, in conjunction with the soldier's office, grade, rank, rating, and performance as recorded by his commander on enlisted/officer evaluation reports and personnel statements of work performance. The PEB is composed of two line officers and a physician, whereas the MEB is composed of physicians. Legal counsel provided to the soldier is independent of the PEB.

Soldiers are referred to the PEB through the PEBLO. The PEBLO is the linchpin in the army

EXHIBIT 16-1

PHYSICAL EVALUATION BOARD: WEIGHING THE EVIDENCE

Objective	vs	Subjective
Acute	vs	Chronic/Long-term
Medical	vs	Performance Administrative

Acute or Grave	Chronic/Long-term
Medical data generally	Performance
More important	Administrative

MEB: Medical Evaluation Board
PDB: Physical Disability Board
PEB: Physical Evaluation Board

Fig. 16-6. The soldier can agree or disagree with the informal board determination of fitness. If he agrees, the board is sent to the Physical Disability Board (PDB) for administrative implementation. If he disagrees, a formal Physical Evaluation Board (PEB) is convened.

physical disability system—the point of contact for the MEB, the soldier, and the PEB. The PEBLO counsels the soldier regarding the results of the MEB NARSUM, assembles all pertinent records, and forwards the MEB NARSUM with appropriate records (the MEB packet) to the PEB in the most expeditious manner available. In the army, the PEBLO is appointed by and works for the MTF Commander. The duties of the PEBLO are set forth in AR 635-40.[22(AppC,§1,p67)]

The PEB Process

In the following discussion of the PEB process, a typical uncomplicated active duty soldier will be presented. Various alternative considerations will be presented later. At every step of the process the soldier is afforded due process and a full and fair hearing. The PEB is a proponent for the soldier as well as the government.

After it has been signed by the approving authority and the soldier, the MEB packet (the MEB NARSUM, pertinent records, and performance data) is sent from the PEBLO to the PEB. On receipt, it is logged in by a case analyst and rechecked to ensure that all necessary paperwork is present and in order. The case then undergoes an informal review that results in a finding of fit or unfit (Figure 16-6).

If determined to be fit, the soldier is returned to duty. If determined to be unfit, a disability percentage is assigned to the soldier on the basis of the VASRD. Because the VASRD was originally written in 1946, it does not include many current medical diagnoses, a problem that is presently being corrected by the preparation of a revised and more up-to-date version. When a diagnosis in the MEB NARSUM is not in the VASRD, the PEB must select a closely related listed illness and rate the soldier accordingly. This process is known as "rating by analogy" (Table 16-1 contains some examples of analogous ratings). The physician, by using the VASRD, affords the PEB the best chance of reaching the right rating regarding the soldier's illness. Examples of disability ratings are shown in Table 16-2.

Disability equates to economic loss, for which the individual is compensated. Ideally, the compensation should reflect the average loss of the soldier's earning capacity as a civilian. However, the army compensates the soldier for only the medical condition or conditions that make him unfit for military duty. The Veterans Administration compensates the soldier for all service-connected medical conditions that would have an impact on civilian employment. This is the basis for the percentage ratings contained in the VASRD. If the disability rating is less than 30%, the soldier is separated with severance pay. If the disability rating is greater than 30% and the disease process is stable, permanent disability retirement is awarded.

The informal review can also come to the conclusion that the disease process is not stabilized to the point where permanent disposition can be made. Accordingly, the soldier may be placed on the TDRL. The requirements for placement on the TDRL are the same as for permanent retirement for medical disability. The soldier must be unfit, must

TABLE 16-1

ANALOGOUS RATINGS*

Illness	VASRD Code	Analogous Illness
Rhabdomyolysis	5099-5021	Myositis
A-C Separation	5299-5003	Arthritis (pain)
Carpal, Bone Injury	5299-5212	Impairment of radius
Anterior Compartment Syndrome	5299-5312-8723	Muscle injury/Deep peroneal nerve
Pilonidal Cyst	7899-7806	Eczema
Thoracic Outlet Syndrome	8599-8513	All radicular nerve
Tension Headaches, Psychogenic	9499-9423	Undifferentiated somatoform disorder
Hypercoagulable States Rate residuals	7199-7120-7121	Varicose veins/Phlebitis
Ileostomy/Colostomy	7399-7330	Intestinal fistula
Hematological Malignancies	7799-7700	Anemia, pernicious
Stroke	9399-9300	Organic mental disorder plus residuals
Hemiplegia	8599-8520 8513	Sciatic nerve All radicular groups, one side
Paraplegia	8599-8520	Sciatic nerve, bilateral factor
Quadriplegia	8599-8520 8513	Sciatic nerve All radicular groups, bilateral factor

VASRD: Veterans Administration Schedule for Rating Disabilities
*Consult Army Regulation 635-40, Appendix B for other guidance

have a disability rating of at least 30%, or must have at least 20 years of service and be eligible for retirement. The medical condition must be temporary or unstable. A soldier who is determined to be fit will not be placed on the TDRL. By law a soldier may not remain on TDRL for more than 5 years. Termination of TDRL status may occur at any time prior to the 5-year maximum. At the end of 5 years, one of the following determinations must be made. The soldier is (1) retired permanently, (2) separated with severance pay, or (3) found fit. TDRL pay and entitlements automatically stop after 5 years unless the case had been finalized by one of the three prior findings, or variations thereof.

When the soldier is informed of the PEB's findings by the PEBLO, he has 10 days to agree or to disagree with the informal findings. If the soldier agrees and is fit, he is returned to duty. If he agrees and is found unfit, he is processed for separation or retirement. The informal board resolves about 75% of the PEB's cases. If the soldier disagrees with the findings of the informal board, he may request (1) a formal hearing with or without a personal appearance or (2) waiver of a formal hearing with or without a written rebuttal. The soldier may request representation by appointed military legal counsel or by civilian counsel at his own expense. An appointed military lawyer representing a soldier does so with the best interest of the soldier foremost. A soldier who is found fit cannot request a formal board but may file a written rebuttal.

A formal board is convened when (*a*) the soldier or next of kin/guardian requests a formal board or (*b*) the PEB president decides it is in the best interest of the soldier and the army.[22(ch4,¶21a,p15)]

Although a formal board has the same composition as an informal board, the specific members may differ (Figure 16-7). The president of the PEB will establish the date, time, and place of the hearing, providing the soldier with a minimum of 3 working days in which to prepare his/her case.[22(ch4,¶21d)] The PEB will notify the soldier of the

TABLE 16-2

VETERANS ADMINISTRATION SCHEDULE FOR RATING DISABILITIES (VASRD)

Code	Medical Impairment and Rating	%
5292	Spine, limitation of motion of, lumbar:	
	Severe	40
	Moderate	20
	Slight	10
5293	Intervertebral disc syndrome:	
	Pronounced; with persistent symptoms compatible with sciatic neuropathy with characteristic pain and demonstrable muscle spasm, absent ankle jerk, or other neurological findings appropriate to side to diseased disc, little intermittent relief	60
	Severe; recurring attacks, with intermittent relief	40
	Moderate; recurring attacks	20
	Mild; recurring attacks	10
	Postoperative, cured	0
5294	Sacroiliac injury and weakness	
5295	Lumbosacral strain:	
	Severe; with listing of whole spine to opposite side, positive Goldthwaite's sign, marked limitation of forward bending in standing position, loss of lateral motion with osteoarthritic changes, or narrowing or irregularity of joint space, on some of the above with abnormal mobility on forced motion	40
	With muscle spasm on extreme forward bending, loss of lateral spine motion; unilateral, in standing position	20
	With characteristic pain on motion	10
	With slight subjective symptoms only	0

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scheduled hearing, notify board members and support staff, arrange for attendance of all military witnesses or obtain depositions, ensure that the soldier's records are furnished to medical witnesses for review before the hearing, and present available witnesses and evidence to the board.[22(ch4,¶d5)] At the formal board the soldier has legal representation, (military, civilian) or self-representation. The appointed military counsel will remain at the hearing and act as co-counsel unless excused by the soldier.[22(ch4.¶e–h)] The duties of the counsel are to safeguard the legal rights of the soldier, prepare the soldier's case for presentation to the board, request that the PEB arrange for witnesses or depositions, examine and cross-examine witnesses, and submit oral and written arguments. The soldier has the right to testify as a witness under oath but also has the right to remain silent.

The proceedings of the PEB are administrative and not judicial in nature; therefore, the board is not bound by rules of evidence prescribed for trials by courts-martial or for court proceedings in general.[22(ch4,¶m.(1))]

The usual formal board begins with the three-member board seated in the hearing room. The soldier, with counsel, reports to the president of the PEB. The president convenes the board and asks the soldier if there has been adequate time for the preparation his case.

Following an affirmative reply, the recorder announces the names and ranks of the board members. At this point the soldier may present any challenges as to board membership and composition. If the soldier challenges a member of the board, and the challenge is upheld by the president of the PEB, that member is excused and replaced by another member who is qualified to sit on the board. If there are no challenges, the president will inquire of the members of the board if they have reviewed the records of the case and whether counsel has any new documentary evidence to present. Having reviewed the new evidence, the president will ask the

NG: National Guard
PDB: Physical Disability Branch
USAPDA: United States Army Physical Disability Agency

Fig. 16-7. The structure of the formal Physical Evaluation Board (PEB) hearing process.

soldier again if he has had adequate time to prepare for the case and if elected counsel is still the soldier's choice. If the response is affirmative, counsel will be asked to present the case. The soldier and any witnesses may be sworn in after initial presentation by counsel or they may elect not to be sworn. Following the swearing in the board members may question the soldier and witnesses. The counsel then presents a summation. Following summation the hearing is closed for deliberation. The meeting is then reopened, the findings and recommendations are presented to the soldier, and the hearing is closed. Following the hearing, counsel discusses the findings with the soldier. The soldier is given a copy of the findings and may elect to make a selection at that time or take up to 10 days to submit a formal rebuttal. On request, counsel will assist in the rebuttal. A formal rebuttal must be based on

1. the decision of the PEB was based on fraud, collusion, or mistake of law;
2. the soldier did not receive a full or fair hearing; and/or
3. substantial new evidence has been obtained that could not be presented prior to disposition by the PEB.[22(ch4,¶21t.(1))]

The evidence on which the rebuttal is based must accompany the rebuttal. Having received a timely rebuttal, the PEB will normally respond within 3 days. The PEB can inform the soldier that (*a*) the rebuttal has been received and has not influenced the outcome of the case but that it will be reviewed by the USAPDA, or (*b*) the PEB has determined that the new information may influence the case. If the latter is true, a formal reconsideration is undertaken, the case is recalled, and the soldier is informed of the outcome of the reconsideration. The soldier has the right to rebut the new findings. The case, along with any rebuttal, will be reviewed by the USAPDA.

The decision options of the formal board are the same as those previously mentioned for the informal board (fit; unfit separated with severence pay [SWSP]; permanent retired [TDRL]). In addition to these most frequently arrived at findings, there are less frequent findings that either the informal or formal board might render.

Soldiers may have medical conditions that existed prior to entry on active duty.[22(ch4,¶19e)] These may present a decided risk to the soldier and to the army. In such cases, if the condition is detected within 6 months of initial entry, and it has been determined that there has been no permanent service aggravation, the soldier may be discharged as personnel who did not meet procurement medical fitness standards.[27(ch2,¶5-11)] In these cases, accession standards,[24(ch12)] rather than retention standards,[24(ch3)] will be applied.

In a similar manner, a soldier with a preexisting disease, usually congenital or hereditary, may be found unfit and separated without benefits (SWOB) if the progression of the disease process is well documented in medical literature and the condition was not permanently service aggravated above and beyond natural progression.

Certain presumptions apply to the physical disability evaluation.[22(ch3,¶2a)] A soldier is presumed to be of sound physical and mental health except for conditions, other than congenital or hereditary, that were noted on the induction physical examination. Any disease or injury discovered subsequently, not due to the soldier's intentional misconduct or willful neglect, is presumed to be service incurred or service aggravated, unless the preponderance of evidence (not merely personal opinion, speculation, or conjuncture) demonstrates otherwise. If a soldier is being processed for separation or retirement for reasons other than physical disability, continued performance of duty commensurate with office, rank, grade, or rating up until the soldier is to be separated or retired, creates a presumption of fitness. This presumption may be overcome if the medical condition actually rendered the soldier unable to adequately perform duties of office, rank, grade, or rating, or if an acute, grave illness occurred immediately prior to or coincident with separation or retirement.[22(ch3,¶2b)]

In certain instances where injury occurred due to the soldier's own misconduct or willful neglect (ie, not in LOD), the soldier is not eligible for benefits.[22(ch4,¶19g)] Thus, it is important that an LOD investigation report accompany the MEB packet when it is submitted to the PEB. Delay in submission of the LOD investigation report may be the single most important factor in delaying the processing of boards.

In cases of Army Reserve soldiers who are on continuous active duty for more than 30 days, the same rules apply as those for active duty soldiers. For Reserve soldiers who are on active duty less than 30 days, proximate result must be shown. That is, causal relationship must be established between the disability and the required military duty. An LOD determination must be accomplished for those cases. Regulations regarding Reserve Component soldiers are contained in AR 40-501[24(ch9,p66)] and AR 635-40[22(ch8,p43)]

If the PEB finds a soldier unfit, the percentage of disability determines whether the soldier is separated with severance pay; permanently retired; or, if the condition is unstable, temporarily retired. However, the PEB may find that although the soldier is unfit, he should be separated without benefits because (*a*) of LOD findings; (*b*) of existence of disease prior to service that was not permanently service aggravated; or (*c*) proximate cause was not established. The soldier has the right to agree or disagree with the findings. All disagreements are forwarded to USAPDA for review (Figure 16-8).

Review by the USAPDA

In addition to cases in which the soldier disagrees with the findings of the PEB, the USAPDA reviews cases (*a*) of all general officers and medical corps officers, (*b*) in which a voting member of the PEB submits a minority report, (*c*) that have previously been forwarded to the USAPDA and that were returned to the PEB for reconsideration, (*d*) designated by the Commanding General, (*e*) of soldiers assigned to the Disability System (PEB or USAPDA), (*f*) of special interest, and (*g*) selected for quality review.[22(ch4,§V,¶22)]

The USAPDA review is confined to the case records, proceedings, and related evidence. Oversight responsibility lies with the USAPDA to ensure that (*a*) the soldier received a full and fair hearing; (*b*) the proceedings of the MEB and PEB were conducted according to governing regulations; (*c*) the findings and recommendations of the MEB and PEB were just, equitable, consistent with facts, and in keeping with provisions of law and regulations; (*d*) due consideration was given to rebuttals submitted to the PEB; and (*e*) records of the case are accurate and complete.

A case analyst logs in each case on receipt at the USAPDA and distributes it to the agency physician, who reviews the case for medical completeness and agrees or disagrees with the PEB findings. If the physician agrees, the case is forwarded to the Personnel Management Officer (PMO); if the physician, however, disagrees, he prepares a proposed Modification (MOD) to the findings and calls the PEB to discuss the case with the PEB physician. This discussion is annotated, and if there is no mitigating evidence for the findings by the PEB, the proposed MOD is forwarded to the PMO.

The PMO reviews the case with special emphasis on how the medical condition relates to the soldier's assigned duties, office, rank, evaluation reports, and commander's statement of performance. Personnel records are again screened to ensure that administrative or judicial actions have not been taken by the soldier's unit or the Department of the Army. Reserve and National Guard cases are reviewed to ensure that they are eligible for disability processing. The PMO is usually a Reserve or National Guard member.

If the PMO determines that there is a discrepancy on any of these issues, a MOD may be generated by the PMO. The PMO also reviews any MOD generated by the physician and agrees or disagrees with the MOD. If there is disagreement, the PMO discusses the case with the physician and consensus is reached. The case is then reviewed by the Operations, Evaluations, and Analysis Officer (OEA). The OEA reviews all cases previously listed plus any MODs, and prepares the cases for signing by the Deputy Commander. All cases with potential legal ramifications are reviewed by USAPDA legal counsel.

Final Outcome of the Review by USAPDA

Any case in which USAPDA agrees with the PEB is forwarded to the PDB for the issuance of orders. Any case with a MOD is returned to the PEB for information. The soldier is informed of that MOD by the USAPDA. The soldier may submit a written rebuttal if he disagrees with the MOD; if he does,

MEB: Medical Evaluation Board
MOD: modification
PDB: Physical Disability Branch
PEB: Physical Evaluation Board

Fig. 16-8. The overall structure of the complete US Army physical disability process.

the case is again reviewed with the rebuttal. USAPDA informs the soldier whether the rebuttal was upheld (agreed with) and the findings changed, or whether the findings were unchanged. If the rebuttal is not upheld, the case is forwarded to the Army Physical Disability Appeals Board (APDAB) for review. APDAB is one of three independent boards of review that advise the Secretary of the Army; it is not part of USAPDA. If APDAB agrees with USAPDA, the case is finalized and the soldier is either discharged, separated, or returned to duty. If APDAB disagrees, the case is reviewed again by the USAPDA and the appropriate action taken as recommended by APDAB.

In summary, the USAPDA may (*a*) agree with the findings of the PEB; (*b*) return the case to the PEB

TABLE 16-3

KEY STATUTORY PROVISIONS OF THE CAREER COMPENSATION ACT OF 1949, U.S.C. 63 STAT. 802 AS AMENDED 10 U.S.C. 1201 ET SEQ

Disposition	LOD	Entitled To Basic Pay	Proximate Result*	Severity > 30%	Severity < 30%
Permanent Disability Retired List	Yes	Yes	Yes	Yes†	No
Temporary Disability Retired List‡	Yes	Yes	Yes	Yes†	No
Separate With Severance Pay	Yes	Yes	Yes	NA	Yes
Separate Without Benefits	No	No	No	NA	NA

LOD: line of duty
*Reserve component soldiers who have served 30 days or less on active duty
†If otherwise retirement eligible, any percentage
‡5-year limit by law

for reconsideration when case records indicate that such reconsideration is in the best interest of the soldier and the government; or (*c*) issue revised findings, that is, modify the disposition or rating of the soldier. In such cases, a detailed explanation will accompany the change. The final appeal determination that USAPDA can grant is referral of the case to APDAB.

When findings are revised, USAPDA will (*a*) furnish the soldier a copy of the revised findings with information copies to the PEBLO and the soldier's counsel; (*b*) advise the soldier he has 10 days to accept or rebut the revision in writing; or (*c*) if the soldier has not had a formal hearing, return the case to the PEB, recommending a formal hearing.

If the soldier agrees with the revision, it will be approved for the Secretary of the Army and forwarded to the PDB for orders. If the soldier disagrees with the review and submits a statement of rebuttal, and if consideration of the rebuttal does not result in any change, a letter will be forwarded to the soldier indicating that no change was made and the case will then be forwarded to APDAB for review. This review will be the final review and the recommendation by APDAB will be returned to the USAPDA who, after making the recommended changes, will forward the case to the Total Army PERSCOM for final disposition. PERSCOM makes final disposition of the cases based on the final decision of USAPDA or APDAB. Orders vary with the final outcome of the case: (*a*) permanent retirement for physical disability (10 U.S.C. 1201 or 1204); (*b*) placement on the TDRL (10 U.S.C. 1202 or 1205); (*c*) separate for physical disability with severance pay (10 U.S.C. 1202 or 1206); (*d*) separate for physical disability without severance pay (sections 630, 1162(a), 1165, or 1169, Title 10 United States Code; (*e*) transfer of soldier who had 20 qualifying years in the reserve to inactive reserve on soldier's request (10 U.S.C. 1209); (*f*) release from active duty and return to retired status for retired soldier serving on active duty who was found to be physically unfit; or (*g*) return to duty of soldiers found fit. This process is summarized in Table 16-3. If the soldier still does not agree with the disposition, he may petition the Army Board for Correction of Military Records requesting review and change when he is no longer in the military.

Statistics Illustrating the Function of the Army Physical Disability System

The following statistical data are presented to illustrate the volume and variety of dispositions made by the army physical disability system. The overall workload of the USAPDA since 1987 has been relatively constant except for the peaks in the years 1991 and 1992 that reflect the effect of the army mobilization during the Persian Gulf War (Figure 16-9). During the period FY 1987 through FY 1997, on average, about 88% of the new cases were active duty soldiers and 12% reserve component. During the first half of this period there were four PEBs (the Georgia PEB closed in 1993). The partition of the caseload among the three existing PEBs was relatively similar during FY 1994 through FY 1997: Walter Reed Army Medical Center processed 35% of the cases, Fort Sam Houston 36%, and Fort Lewis the other 29% (Table 16-4). It should be noted that at this time, there is not an exact parallel configuration between the largest 15 MTFs and the three PEBs (Table 16-5).

TABLE 16-4

PHYSICAL EVALUATION BOARDS CASE DISTRIBUTION: TOTAL CASES RECEIVED

Fiscal Year	Walter Reed Army Medical Center Washington, DC	Fort Gordon Georgia*	Fort Sam Houston Texas	Fort Lewis Washington
1989	2,536	2,150	2,592	2,567
1990	2,592	2,725	3,258	2,732
1991	3,348	2,876	3,578	2,896
1992	2,540	3,373	3,776	2,933
1993	1,986	1,458	1,761	1,388
1994	2,562		2,774	2,266
1995	2,542		2,582	2,249
1996	2,979		2,601	2,408
1997	2,660		3,296	2,164
TOTALS	23,745	12,582	26,218	21,603

*Georgia Physical Evaluation Board closed in 1993

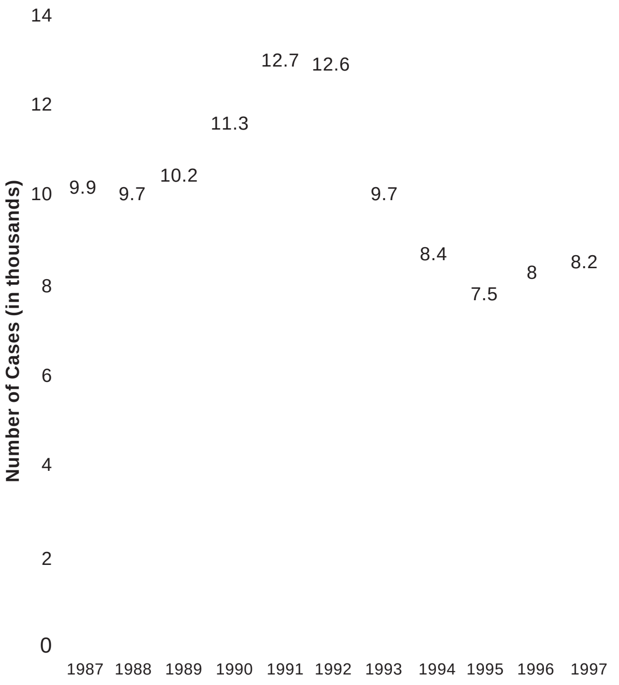


Fig. 16-9. US Army physical disability system, overall work load completed for fiscal years 1987 through 1997.

TABLE 16-5

TOP 15 POSTS SENDING CASES TO THE DC, TEXAS, AND WASHINGTON PEBs (Total Combined Cases Sent: 5,066)

MTF	Cases	PEB Processing Cases
Walter Reed AMC	618	WRAMC, Washington, DC
Fort Campbell	432	Fort Sam Houston, Texas
Fort Hood	411	Fort Sam Houston, Texas
Fort Knox	387	Fort Sam Houston, Texas
Fort Sam Houston	340	Fort Sam Houston, Texas
Fort Gordon	340	Fort Sam Houston, Texas
Fort Bragg	338	WRAMC, Washington, DC
Tripler AMC	309	Fort Lewis, Washington
Fort Benning	300	Fort Sam Houston, Texas
Fort Stewart	292	Fort Sam Houston, Texas
Madigan AMC	289	Fort Lewis, Washington
Fort Leonard Wood	280	Fort Lewis, Washington
Fort Polk	252	Fort Lewis, Washington
Fort Carson	247	Fort Lewis, Washington
Fitzsimons AMC	231	Fort Lewis, Washington

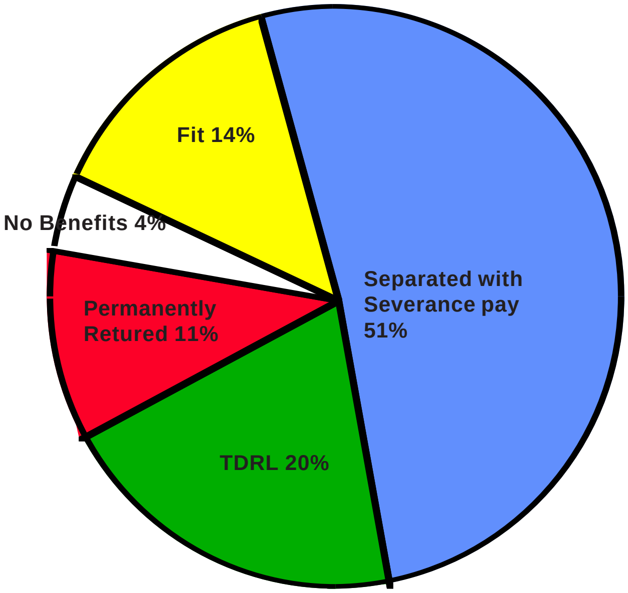
AMC: Army Medical Center
MTF: Medical Treatment Facility
PEB: Physical Evaluation Board
WRAMC: Walter Reed Army Medical Center

TABLE 16-6

MOST FREQUENTLY USED VASRD CODES

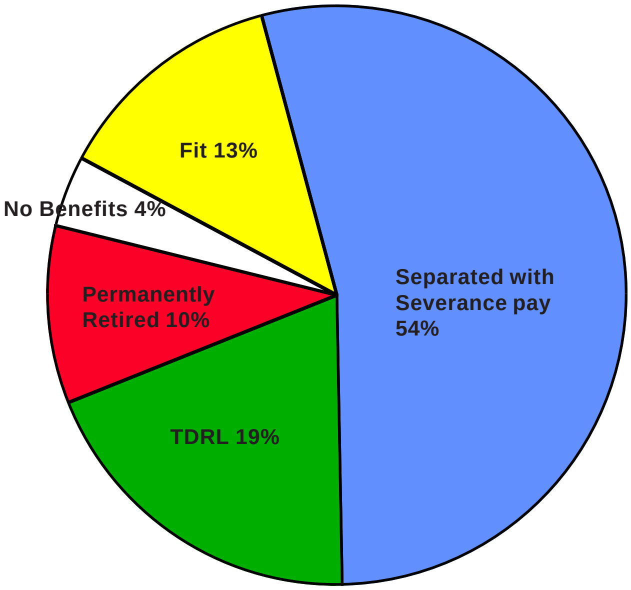
VASRD	Cases	Total Cases %
5003	Arthritis (degenerative)	11.9
5295	Back (physical)	8.3
5257	Knee	6.0
5293	Back (neurologic)	2.9
5010	Arthritis (traumatic)	2.3
6351	HIV	2.2
9208	Bipolar Disorder	2.2
9207	Major Depression	2.2
8045	Brain Disease	1.5
6602	Asthma	1.4
5255	Femur	1.3
5024	Tenosynovitis	1.2
9210	Psychosis	1.2
9304	Dementia	1.2
5276	Flat Feet	1.2

HIV: human immunodeficiency virus
VASRD: Veterans Administration Schedule for Rating Disabilities



TDRL: Temporary Disability Retired List

Fig. 16-10. Total Physical Evaluation Board case dispositions for fiscal years 1987 through 1997, by outcome (108,200).

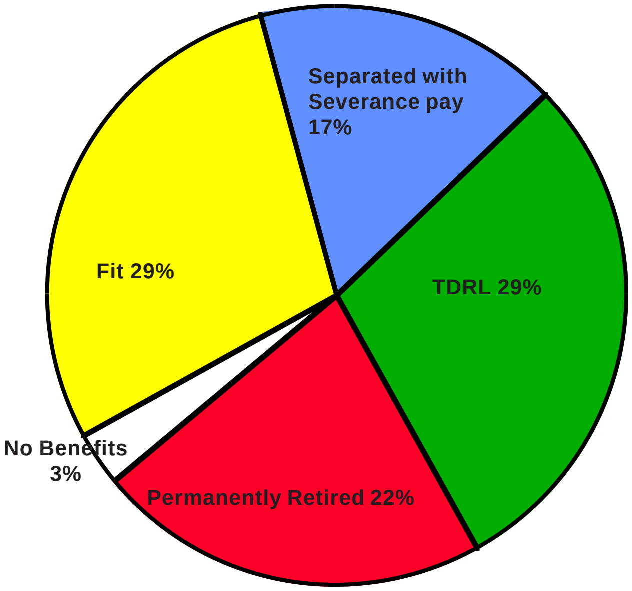


TDRL: Temporary Disability Retired List

Fig. 16-11. Total Physical Evaluation Board case dispositions of enlisted personnel for fiscal years 1987 through 1997, by outcome (99,544).

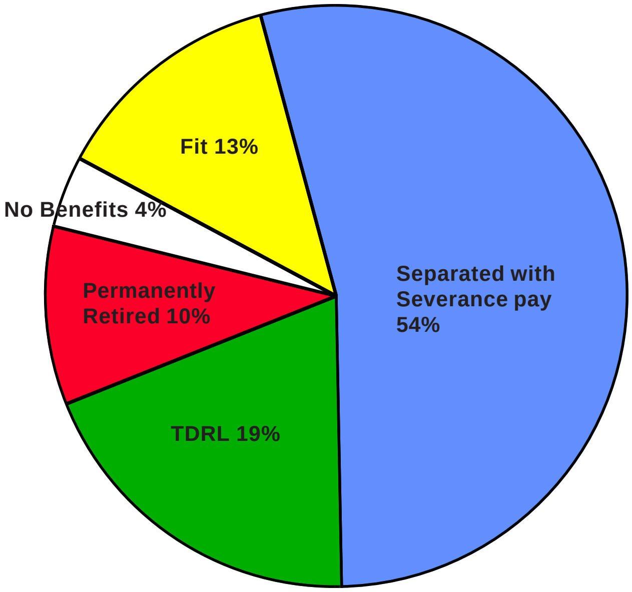
Soldiers entering the army physical disability program are found to have a large variety of medical conditions, with most occurring with low frequency. The 15 most common medical conditions coded by the VASRD account for only 47% of the total number of cases (Table 16-6). These codes will change because of recent policy changes; many of the 5003 codes for arthritis are being moved to the exact anatomical location, most commonly 5295 (back) or 5257 (knee). In addition, all human immunodeficiency virus (HIV) cases are being coded as 6351. Prior to 1991, codes 6351, 6352, and 6353 were all used for HIV.

The disposition outcomes of cases by PEBs fall into five categories: (1) separation with severance pay, (2) placement on the temporary disability retired list, (3) permanently retired, (4) separated without benefits, and (5) found fit. When all dispositions are considered for the period FY 1987 through FY 1997, the most common outcome was separation with severance pay (51%) (Figure 16-10). Almost one-fifth of cases saw no award of money because most soldiers in this category were either found fit or separated without benefits. The disposition for enlisted men is shown in Figure 16-11; the



TDRL: Temporary Disability Retired List

Fig. 16-12. Total Physical Evaluation Board case dispositions of officers for fiscal years 1987 through 1997, by outcome (8,656).

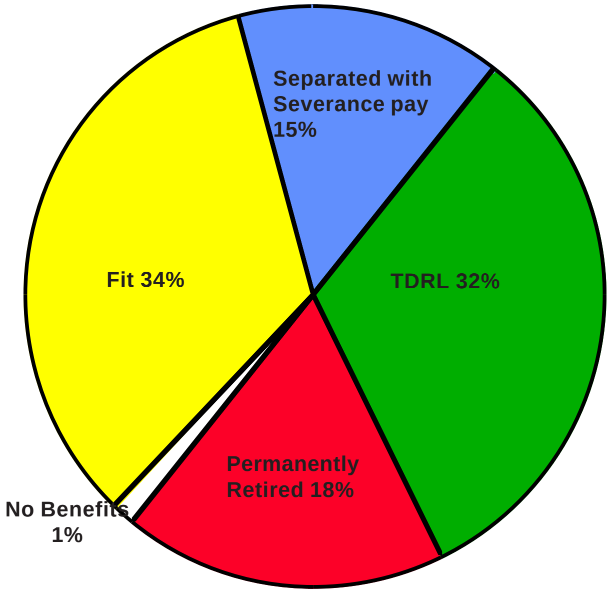


TDRL: Temporary Disability Retired List

Fig. 16-13. Total Physical Evaluation Board Regular Army case dispositions for fiscal years 1987 through 1997, by outcome (95,216).

disposition for officers is shown in Figure 16-12. Compared to enlisted personnel, officers were more than twice as likely either to be found fit or to be permanently retired. Conversely, it was uncommon for officers to be separated with severance pay. In considering these data it should be borne in mind that the officers were older and were more likely to have multiple diagnoses that were of a chronic nature. Enlisted soldiers had more acute injury residuals and were younger, usually being healthy otherwise. It is to be expected that the increased number of diagnoses and the increased severity of the disease in officers resulted in a higher overall disability rating reflected in the higher percentage permanently retired.

At any given time, members of the Total Army consist of three components: Active Army, the Army National Guard (ANG), and the Army Reserve (USAR). The Active Army, the principle source of cases for the army physical disability system, consists of the Regular Army, soldiers of the National Guard on active duty, and activated members of the Reserve. The PEB disposition of Regular Army and Active Guard Reserve (AGR) soldiers on active duty might be expected to differ from soldiers who have been temporarily activated (Figures 16-13, 16-14, and 16-15). Regular Army and AGR soldiers have a similar distribution of dispositions, but compared to temporarily activated soldiers seen by the PEBs, have a much lower probability of being found fit



TDRL: Temporary Disability Retired List

Fig. 16-14. Total Physical Evaluation Board Army National Guard case dispositions for fiscal years 1987 through 1997, by outcome (5,410).

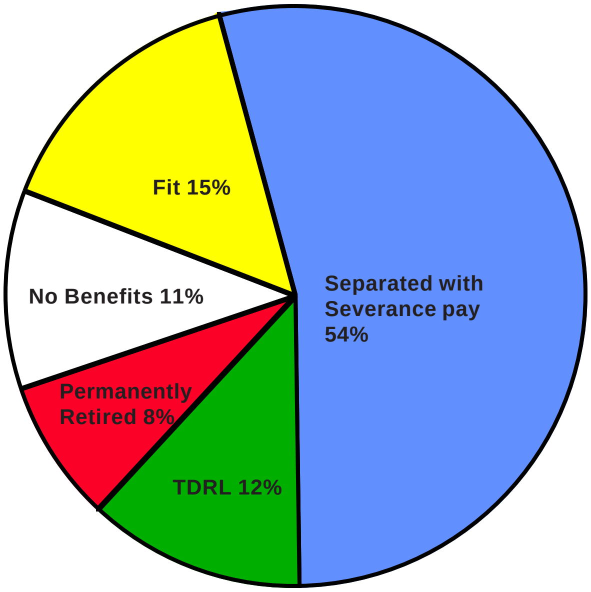


Fig. 16-15. Total Physical Evaluation Board Army Reserve case dispositions for fiscal years 1987 through 1997, by outcome (7,574).

or being placed on the TDRL. Conversely, Reserve and National Guard soldiers on temporary active duty are less likely to be separated with severance pay. One way of interpreting the finding is that a larger percentage of activated National Guard and Reserve component soldiers are being found fit because these soldiers wanted to be found fit, a conclusion supported by their performance data, including their commanders' statements and evaluation reports. Analysis of the increased percentage of SWOBs for the Reserve Component indicated a direct correlation with the proximate result findings (ie, a direct result of performing military duty). In the majority of these cases the medical impairment was not the direct result of performance of military duty. The PEB is a performance based system and the Commander knows best how his soldiers can perform assigned tasks.

Benefits paid by the USAPDS for FY 1987 through 1989 averaged $477 million per fiscal year. This amount has been fairly constant year by year except for FY 1990 through FY 1992, which reflects the Persian Gulf War years. Data for FY 1997 indicate that $344.5 million was paid out in benefits; 76% went to soldiers who were permanently retired; 18.8% went to soldiers who were separated with severance pay; and 5% of the money went to soldiers who were placed on the TDRL, even though 20% of total dispositions in FY 1987 through FY 1997 were TDRL. Thus, it was more cost-effective from the government's standpoint to place soldiers on TDRL rather than to permanently retire them when their disease/condition first manifests itself.

THE US NAVY AND AIR FORCE DISABILITY SYSTEMS

The following is a brief review of the US Navy and Air Force disability systems and a comparison of disability dispositions for the three services for FY 1997 (Table 16-7). There is no separate marine disability system; the navy disability system is also responsible for the marines.

US Navy

The naval disability system is a component of the Naval Council of Personnel Boards and is directly under the Assistant Secretary of the Navy for Manpower and Reserve Affairs. The navy has one informal PEB and two formal PEBs. The formal boards are located in San Diego, California, and at the National Naval Medical Center, in Bethesda, Maryland. Although different individuals sit on the informal and formal boards, each of the boards is composed of one navy line officer, one marine line officer, and one navy physician.

In FY 1997 the navy processed 10,196 cases; 1,057 (10%) sailors and marines were found fit; and 9,139

TABLE 16-7

TRISERVICE COMPARISON STATISTICS FY 1997

Element	Air Force	Army	Navy
Total New Cases*	4,045	8,038	10,196
Fit	1,776	1,162	1,057
Unfit	2,269	6,876	9,139
SWSP	867	4,109	5,258
PERM	648	850	1,071
TDRL	655	1,584	2,045
SWOB	99	333	765

PERM: Permanent Disability Retired List
SWOB: separated without benefits
SWSP: separated with severance pay
TDRL: Temporary Disability Retired List
*Old TDRL cases not counted

(90%) were found unfit. Of the unfit sailors and marines, 5,258 (58%) were separated with severance pay; 1,071 (12%) were permanently retired; 2,045 (22%) were placed on the TDRL; and 765 (8%) were separated without benefits.

US Air Force

The Air Force disability system has one informal board and one formal board. Both of these boards are part of the Air Force Personnel Center located at Randolph Air Force Base, Texas. The Informal Board is composed of two air force physicians and one air force line officer; the Formal Board has two air force line officers and one air force physician. The members sitting on the informal and formal boards are not the same individuals. In FY 1997 the air force processed 4,045 airmen, of whom 1,776 (44%) were found fit and 2,269 (56%) were found unfit. Of those airmen found unfit, 867 (38%) were separated with severance pay; 648 (29%) were permanently retired; 655 (29%) were placed on the TDRL; and 99 (4%) were separated without benefits.

Comments

As one can see, the Disability Boards of the three services are fairly consistent in the findings of service members appearing before them. This is to be expected because they are all governed by the same law (Chapter 61, Title 10 U.S.C.). The differences can be explained in the manner in which the service secretaries choose to implement the disability system within their service. The army has the same members sitting on their informal and formal boards. The air force and the navy have different members sitting on their informal and formal boards. The air force finds a greater percentage of airmen fit due to the fact that they have within each of their units a given number of nondeployable slots to which airmen may be assigned. With these slots being available, the air force board can find an airman fit and assign him to a nondeployable slot. As a group these individuals are classified as Code C. The army and navy do not have a comparable classification and, thus, have a lower percentage of fit findings.

CONCLUSION

The United States Army Physical Disability System is a segment of the army unknown to the majority of soldiers—those who have never had significant medical conditions. But to that minority whose military careers have been terminated by medical conditions, it becomes an advocate for transition of the disabled soldier into civilian life. The disability system assists the soldier at every stage with counseling to ensure that he receives a full and fair hearing and just compensation for a medical condition sustained while in service of his country.

For over 50 years, the present disability system has been helping soldiers. It is an integral part of the army in the maintenance of a full and fit force. For soldiers who cannot continue military service due to medical impairment, the army stands ready to provide for them through a "Quality Disability System Administered with Pride."

ACKNOWLEDGMENT

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ACRONYMS AND ABBREVIATIONS
Volumes 1 and 2

A

AAEM: American Association of Electrodiagnostic Medicine
AAROM: active assistive range-of-motion
ABG: arterial blood gas
ABPM&R: American Board of Physical Medicine and Rehabilitation
ABPM: American Board of Physical Medicine
ABR: auditory brainstem response
AC: acromioclavicular
ACE: angiotensin converting enzyme
ACL: anterior cruciate ligament
ACTH: adrenocorticotropic hormones
ADH: antidisseretic hormone
ADL: activities of daily living
ADP: adenosine diphosphate
AE: above elbow
AFO: ankle/foot orthosis
AFQT: Armed Forces Qualification Test
AGR: Active Guard Reserve
AKA: above-knee amputation
AMC: Army Medical Center
AMEDD: U.S. Army Medical Department
AMSC-CIR: Army Medical Specialist Corps Clinical Investigation and Research
AMSC: Army Medical Specialist Corps
ANG: Army National Guard
AP: anterior–posterior
APDAB: Army Physical Disability Appeals Board
AR: Army Regulation
ARC: American Red Cross
ARG: Army Reservists and National Guard
AROM: active range-of-motion
ARVN: Army of the Republic of Vietnam
ASIA: American Spinal Injury Association
ATH: air transportable hospital
ATN: acute tubular necrosis
ATP: adenosine triphosphate
ATPase: adenosine triphosphatase

B

BAEP: brainstem auditory evoked potential
BAER: brain stem auditory evoked response
BAM: basilar artery migraine
BE: Below elbow
BEAM: brain electrical activity mapping
BEE: basal energy expenditure
BIA: Brain Injury Association
bid: twice daily
BKA: below-knee amputation
BP: blood pressure
BSA: body surface area
BUN: blood urea nitrogen

C

CAD/CAM: computer aided design/computer aided manufacturing
CAPTE: Commission on Accreditation for Physical Therapy Education
CASD: computer aided socket design
CAT/CAM: contoured adducted trochanteric-controlled alignment method
CDP: cytidene 5′-diphophocholine
CED: cognitive enhancing drug
CK: creatine kinase
CMAP: compound motor action potential
CMRR: common mode rejection ratio
CMST: circulation, motion, sensation, and temperature
CMUAP: compound motor unit action potential
CN: cranial nerve
CNS: central nervous system
C.O.: cardiac output
CO$_2$: carbon dioxide
COMMZ: combat zone
CONUS: continental United States
CP: conventional prosthesis
CP: creatine phosphate
CPAP: continuous positive airway pressure
CPM: continuous passive motion
C-PMN: C polymodal nociceptors
CPR: cardiopulmonary resuscitation
CRPS: Complex Regional Pain Syndrome
CRT: cathode ray tube
CSF: cerebrospinal fluid
CT: computed tomography
CTR: carpal tunnel release
CTS: carpal tunnel syndrome

D

dB: decibel
DCCS: Deputy Commander for Clinical Services
DEXA: dual photon X-ray absorptiometry
DF: dorsiflexion
DIP: distal interphalangeal
diphosphonate EHDP: disodium ethane-1-hydroxy-1, 1-diphosphonate
DoD: Department of Defense
DREZ: dorsal root entry zone
DVA: Department of Veterans Affairs
DVT: deep venous thrombosis

E

ECU: environmental control unit
EEG: electroencephalogram/electroencephalographic/electroencephalography
EF: Epileptiform findings
EMG: electromyographic/electromyography
ENG: electronystagmography
EPB
ERG: electroretinogram
ERMPs: early recovery management programs
ESWL: extracorporeal shock wave lithotripsy

F

FAD: (McMaster) Family Assessment Devise
FAD: flavin adenine dinucleotide
FADH$_2$: FAD gains electrons to become
FAM: Functional Assessment Measure
FDA: Food and Drug Administration
FDP: fibrin degradation product
FES: functional electrical stimulation
FF: full field
FIM: Functional Independence Measure
FNQ: Family Needs Questionnaire

FS: focal slowing
FVC: forced vital capacities
FY: fiscal year

G

GABA: gamma-aminobutyric acid
GAP-43: growth-associated protein-43
GCS: Glasgow Coma Scale
GED: General Education Diploma
GI: gastrointestinal
GMO: general medical officer
GOS: Glasgow Outcome Scale
GPB: glossopharyngeal breathing

H

H_2: Histamine$_2$-receptor
HDL: high density lipoprotein
HF: half field
HIV: human immunodeficiency virus
HO: heterotopic ossification
HR: heart rate
HTL: hearing threshold level
HTM: high threshold mechanoreceptors

I

IASP: International Association for the Study of Pain
IC: intermittent catheterization
ICP: intracranial pressure
ICU: intensive care unit
International 10-20: The International Ten-Twenty System of Electrode Placement
IP: interphalangeal
IPI: interpotential interval
IPOP: immediate postoperative prosthesis
IPORD: immediate postoperative rigid dressing
ISNY: Icelandic-New York
ITB: iliotibial band
IU: international units
IV: intravenous

K

KAFO: knee-ankle-foot orthosis

L

L-dopa: levodopa
LE: lower extremity
LHS: left heelstrike
LLB: long leg brace
LOD: line of duty
LTM: low threshold mechanoreceptors

M

MAOI: monoamine oxidase inhibitor
MCL: medial collateral ligament
MCP: metacarpophalangeal
MCVOST: Medical College of Virginia Olfactory Screening Test
MEB: Medical Evaluation Board
MEDCOM: Medical Command
MEP: motor evoked potential
MESI: Mangled Extremity Syndrome Index
MF2K: Medical Force 2000
ML: medial-lateral
MMPI-2: Minnesota Multiphasic Personality Inventory-2
MMRB: Military Medical Retention Board
MMTN: myelinated mechanothermal nociceptors
MOD: modification
MOS: military occupation specialty
MP: myoelectric prosthesis
MP: metacarpophalangeal
MPA: microstomia prevention appliance
MRI: magnetic resonance imaging
mRNA: messenger ribonucleic acid
MS: multiple sclerosis
MSH: melanocyte stimulating hormone
MTF: medical treatment facility
MTP: metatarsophalangeal
MVC: maximum voluntary isometric contraction

N

NAD^+: nicotinamide adenine-dinucleotide
NADH: NAD+ gains hydrogen to become
NARSUM: narrative summary
NATO: North Atlantic Treaty Organization
NCS: nerve conduction study
NG: National Guard
NGF: nerve growth factor
NHIF: National Head Injury Foundation
NIH: National Institutes of Health
Nm: knee moment
NMDA: *N*-methyl *D*-aspartate
NMS: neuromusculoskeletal
NPO: nothing by mouth
NPT: nocturnal penile tumescence
NSAID: nonsteroidal antiinflammatory drug
NSNA: normal shape, normal alignment

O

OBLA: onset of blood lactate accumulation
OEA: Operations, Evaluations, and Analysis Officer
OH: orthostatic hypotension
OT: occupational therapist/therapy
OTSG: Office of The Surgeon General

P

PA: Physician Assistant
PAFO: plastic ankle-foot orthosis
PCA: patient controlled analgesia
P_{CO_2}: partial pressure of carbon dioxide
PDB: Physical Disability Branch
PEB: Physical Evaluation Board
PEBLO: Physical Evaluation Board Liaison Officer
PEEP: positive end-expiratory pressure
PERSCOM: Personnel Command
PF: plantarflexion
PFT: pulmonary function test
PIP: proximal interphalangeal
PMO: Personnel Management Officer
PMR: physical medicine and rehabilitation
PNF: proprioceptive neuromuscular facilitation
P_{O_2}: partial pressure of oxygen
PROM: passive range-of-motion
PT: physical therapist/therapy
PT: prothrombin time
PTA: posttraumatic amnesia
PTB-TCS: patellar tendon bearing, total contact socket
PTE: posttraumatic epilepsy
PTSD: posttraumatic stress disorder

R

1RM: one repetition maximum test
RDA: recommended dietary allowance
RGO: reciprocating gait orthosis
RH: thyrotropin releasing hormone
RHO: right heeloff
RHS: right heelstrike
RQ: respiratory quotient
RSD: reflex sympathetic dystrophy
RSDS: reflex sympathetic dystrophy syndrome
RTO: right toeoff
RTS: right toestrike

S

3S: silicone suction socket
SACH: solid-ankle cushion-heel
SAFE: stationary attachment, flexible endoskeleton
Sca: Slow component a
Scb: Slow component b
SCI: spinal cord injury
SCL-90: Symptom Checklist-90
SCV: slow component velocity
SEE: standard error of the estimate
SFC: soluble fibrin complex
SIADH: secretion of inappropriate antidiuretic hormone
SIDS: Sudden Infant Death Syndrome
SMP: sympathetically maintained pain
SNAP: sensory nerve action potential
SP: silent period
SPECT: single photon emission computed tomography scans
SPL: sound-pressure level
SSRI: selective serotonin reuptake inhibitors
STEN: STored ENergy foot
STSG: split thickness skin graft
SV: stroke volume
SWOB: separated without benefits
SWSP: separated with severance pay

T

TABPM: American Board of Physical Medicine
TBI: traumatic brain injury
TBSA: total body surface area
TBSAB: total body surface area burned
TCA: tricyclic antidepressants
TCL: tibial collateral ligament
TDRL: Temporary Disability Retired List
TENS: transcutaneous electrical nerve stimulation
TES: total elastic suspension
TD: termal device
THA: tetrahydro-9-aminoacridine
tid: three times per day
TKA: trochanter, knee, ankle (line)
TLE: temperolimbic epilepsy
TLSO: thoracolumbosacral orthosis
TO: Theater of Operations
TOS: thoracic outlet syndrome
TPR: total peripheral resistance
TRH: thyrotropin releasing hormone
TRM: ten repetition maximum
TUN: total urinary nitrogen

U

UCL-BC: University College of London Bioengineering Center
UDS: Utah Dynamic Socket
UE: upper extremity
UN: United Nations
USAPDA: United States Army Physical Disability Agency
USAR: United States Army Reserve
USC: United States Code
UTI: urinary tract infection
UUN: urinary urea nitrogen

V

VA: Veterans Affairs, Department of
VAMC: Veterans Affairs Medical Center
VASRD: Veterans Administration Schedule for Rating Disabilities
VEP: visual evoked potentials
VHIS: Vietnam Head Injury Study
VO_2max: volume of maximum oxygen consumption
VO_2: oxygen consumption

W

WBC: white blood cell
WBGT: wet-bulb globe temperature
WDR: wide dynamic range
WHFO: wrist, hand, finger orthosis
WMSC: Women's Medical Specialist Corps
WRAMC: Walter Reed Army Medical Center

REHABILITATION OF THE INJURED COMBATANT, Volumes 1 and 2

A

AAEM
  See American Association of Electrodiagnostic Medicine (AAEM)
AAROM
  See Active assistive range-of-motion (AAROM)
Abdominal binders
  elastic, 182, 184
Abducens nerve, 291–300
  anatomy and function, 291–293
Abducens neuropathies, 295–296
  electrodiagnosis, 299
  evaluation, 299
  imaging, 299–300
  management, 299–300
  symptoms and signs, 297–299
  syndromes, 296–297
Above-elbow (AE) amputations, 43–44
  long, 44
  short, 44
  surgical procedures, 46
Above-elbow prostheses, 34, 56–59
  versus below-elbow prostheses, 56
  choice of, 69–71
  elbow joints for, 56–57, 59
  fitting, 48–50
  myoelectric, 69
  and residual limb problems, 61
  standard components, 57
Above-knee amputations (AKA), 122–134
  acute pain management, 125
  definitions, 122–123
  energy expenditure for walking, 93
  functional outcome, 83
  interdisciplinary team management case study, 835–837
  joint contracture prevention, 126
  long-term follow-up, 133–134
  low, 82
  mortality rates, 93
  psychological support, 125, 133–134
  rehabilitation, 123–127, 143, 147–148
  reintegration into community, 133
  selection, 123–124
  surgical techniques, 122–124
  unbalanced myodesis in, 140
  weight bearing, 126
Above-knee prostheses
  fitting, 126–127
  training, 133
ABPM
  See American Board of Physical Medicine (ABPM)
ABPM&R
  See American Board of Physical Medicine and Rehabilitation (ABPM&R)
Accessory nerve, 335–338
  anatomy and function, 335–336
Accessory neuropathies, 336–338
  electrodiagnosis, 338
  evaluation, 336–338
  imaging, 338
  management, 338
  prognosis, 338
  symptoms and signs, 336
  syndromes, 336
Accidental death
  incidence, 209–210
Accommodation reflex
  innervation, 295
Ace bandages, 357
Acetazolamide, 217
Acetylcholine, 450, 785
Acetylcholine receptor proliferation
  following denervation, 435–436
Acetylcysteine, 759
Acetylsalicylate
  contraindications, 357
Achilles tendinitis, 399–400
Acid burns, 577–578
AC joint
  See Acromioclavicular (AC) joint
Acknowledgment, 847
ACL
  See Anterior cruciate ligament (ACL)
Acoustic nerve, 315–325
  anatomy and function, 315–317
Acoustic neuroma, 322
Acoustic neuropathies, 317–318
  electrodiagnosis, 322–323
  evaluation, 319–322
  management, 323–324
  prognosis, 323
  symptoms and signs, 318–319
Acromioclavicular (AC) joint, 371–372
  rehabilitation, 372
ACTH
  See Adrenocorticotropic hormone (ACTH)
Actin, 783
Action potentials, 424–425, 785
  duration, 459
  loss of ability to provoke, 460
  measurement, 451
  propagation, 449
  See also Compound motor action potentials (CMAP); Sensory nerve action potential (SNAP)
Active assistive range-of-motion (AAROM)
  for burn patients, 603–606
  definition, 602–603
Activities of daily living (ADL)
  assessment instruments, 852–853
  and bilateral upper extremity amputees, 63–64
  and burn patients, 596, 601, 607, 618–621, 631, 676–680
  limitations, 15
  and lower extremity amputees, 84, 102–104, 106
  and quadruple amputees, 149
  and spinal cord injured patients, 179
  and traumatic brain injuries, 227–228
  and upper extremity amputees, 50–52, 62–64
  See also specific activity
Acupuncture
  for phantom limb pain, 147
Acute abdomen
  in spinal cord injured patients, 184
Acute mountain sickness, 820
Adaptic, 591, 594–595, 600, 616
Adaptive devices
  for bathing, 679
  for burn patients, 618–621, 631, 674–680

  for communication, 620–621, 681–682, 833
  for donning pressure garments, 679–680
  for dressing, 679–680
  for drinking, 619–620
  for driving, 54, 64, 178–179, 682
  for environmental control systems, 621
  for feeding, 619–620, 676–677
  for functional mobility, 682
  for grooming, 620, 677–679
  for hand rehabilitation, 38, 41–42
  for hearing impairments, 324
  for home accessibility, 681
  for housekeeping, 681
  for keyboard usage, 681–682
  for kitchen activities, 681
  for lower extremity amputees, 106
  as orthosis attachments, 707
  for reading, 621
  recreational, 10, 41–42, 52–54, 70–71, 149, 682–683
  for self-feeding, 619–620, 676–677
  for spinal cord injured patients, 178–179
  for toileting, 679
  vocational, 54
  for writing, 621
  See also specific device
Adaptive skills
  assessment, 855
Adenosine diphosphate (ADP), 786
Adenosine triphosphatase (ATPase), 783–784
Adenosine 5′ -triphosphate (ATP), 786–787
  resynthesis, 786–787
Adie's pupil, 297
ADL
  See Activities of daily living (ADL)
Adler-Scheie test, 287
ADP
  See Adenosine diphosphate (ADP)
Adrenocorticotropic hormone (ACTH), 218
Adson maneuver, 501
Advisory Board for Medical Specialties, 5–6, 831
Advocacy
  for brain injured patients, 249, 255
AE amputations
  See Above elbow (AE) amputations
Aerobic capacity
  and age, 804–805
  correlation between body fat percentage and, 795
  and gender, 803–804
  and immobilization, 764
  of military populations, 803–806
  and occupation, 804–805
Aerobic power, 782, 799–801
  definition, 799
  determinants, 800–801
  measurement, 799–800
Aerobic system, 787
Aerobic training, 810–812
  adaptation to resistance training, 812
  adaptive response, 809
  in integrated program, 813–814
  in weight control programs, 793
Affective disorders
  after traumatic brain injury, 223
Afferent fibers
  in cranial nerves, 281
AFO
  See Ankle/foot orthoses (AFO)
African Americans
  counseling concerns, 849
Age
  as risk factor for injury, 816
  and severity of burn injury, 579
  and traumatic brain injury management, 218
  and vocational rehabilitation, 847
  See also Elderly; Children
Ageusia, 311
Air cushion
  three-layered, 757
Air-Flex Foot, 117–119
Air-fluidized bed, 757
Air Force
  body fat standards, 796–798
  disability systems, 884
  physical therapy services, 25–26
Air Force Convalescent Training Program, 831
Airplane splints, 630, 638
Air suspension beds, 757
Airway compromise
  signs of, 583
Airway management
  of burn patients, 583–585, 598–599
Akathisia
  after traumatic brain injury, 220
Alcohol abuse
  and pharmacologic therapy, 240
  and traumatic brain injury, 229, 239–241, 246
Alignment
  dynamic, 104
  static, 103–104, 127
Alkali burns, 577–578
Allergic contact dermatitis
  on residual limb, 139
Allodynia, 482
Allograft skin
  See Homograft skin
Alternate binaural loudness balance test, 320
Altitude
  high terrestrial, 820
Amantadine hydrochloride, 214–215, 217, 219
Amaurotic familial idiocy, 287
Ambulation
  after spinal cord injury, 175, 177–178, 731
  after traumatic brain injury, 226
  by burn patients, 636
  energy expenditure for, 92–93, 149, 178, 723–724, 731, 733
  by fracture patients, 733–737
  impairment, 15
  by lower extremity amputees, 13, 81, 84, 88, 102, 104, 106, 125–127, 135
  by multiple amputees, 148–149
  and musculoskeletal disorders, 358, 384
  See also Crutch ambulation; Gait pattern; Gait training; Gait velocity
American Association of Electrodiagnostic Medicine (AAEM), 11, 448, 513
American Association of Electrotherapy and Radiology, 830
American Association of Physical Therapists, 830
American Board of Medical Specialties, 6
American Board of Physical Medicine (ABPM), 5–6, 831
American Board of Physical Medicine and Rehabilitation (ABPM&R), 6, 832
American Burn Association, 579, 583
American College of Radiology and Physiotherapy, 4
American Medical Association, 5–6, 8
  American Board of Physical Medicine, 5–6, 831
  Council on Physical Therapy, 22

  impairment rating scale, 285
  Special Exhibit Committee on Physical Medicine, 742
American Physical Therapy Association, 20
American Spinal Injury Association (ASIA), 167–169
Americans with Disabilities Act, 6
American Women's Physical Therapeutic Association, 21
Amino acids
  and traumatic brain injury, 212
Amino acid supplementation
  for burn patients, 687
Amitriptyline, 476, 492, 495
Amnesia
  and mild brain injury, 230
  posttraumatic, 253
Amphetamine, 219
Amplification
  electrodiagnostic, 451–452
Amplitude, 459
  motor unit, 457
  nerve conduction studies, 459, 464
Amputation
  Civil War, 34, 71–72, 80, 134
  emotional aspects, 47, 52
  Korean War, 134
  mortality rate, 134
  multiple, 122, 133, 148–149
  Persian Gulf War, 12–15, 46–47, 80
  primary indication for, 45
  psychological trauma, 10
  quadruple, 149
  surgical principles, 45–46
  Vietnam War, 10, 52, 72, 80, 134, 148, 150
  vocational outcomes after, 150
  World War I, 35, 81, 93, 95, 134
  World War II, 34–35, 46, 71–72, 80, 93–95, 134
  See also Lower extremity (LE) amputations; Upper extremity (UE) amputations; *specific type of amputation*
Amputation centers
  World War II, 7, 10, 34–35, 52, 80–81, 94, 149
AMSC
  See Army Medical Specialist Corps (AMSC)
AMSC-CIR
  See Chief of Army Medical Specialist Corps Clinical Investigation and Research (AMSC-CIR)
Anaerobic glycolysis, 787–788
Anaerobic power, 782, 801–802
  definition, 801
  determinants, 802
  measurement, 801–802
  population data, 806
Anaerobic training, 812–813
  in integrated program, 813–814
  See also Resistance training
Anaerobiosis
  muscle, 786, 789
Analgesics
  for burn patients, 599–600, 621–623, 625–626
  for musculoskeletal injuries, 357–358, 383
  for phantom limb pain, 145–146
  for spinal cord injured patients, 191
  See also Pain management; *specific drug*
Anderson shoulder ring, 713
Anesthesia
  for causalgia, 489–490
  for lumbar pain, 382
  malpositioning during, 628
  ocular complications, 296
  for phantom limb pain, 146, 148
  for reflex sympathetic dystrophy, 141, 148
  for residual limb pain, 142, 147
Anesthesia dolorosa, 308
Anesthetics
  for musculoskeletal injuries, 372, 375
  for phantom limb pain, 146
  See also specific drug
ANG
  See Army National Guard (ANG)
Angiography
  pulmonary, 760–761
Aniracetam, 219
Ankle
  musculoskeletal disorders, 395–401
  nerve injuries, 531–545
  scar compression, 675
Ankle compression injury
  chronic or repetitive, 538, 541–542
Ankle disarticulation
  See Syme's amputation
Ankle-foot orthoses (AFOs), 704, 715–726
  ankle joints for, 716–717
  anterior stops, 715–716, 718–720, 723
  biomechanical function, 716–720
  for burn patients, 615, 631, 643
  components, 715–716
  design and suitability, 720–722
  evaluation by biomechanical principles, 722–723
  indications, 716
  mediolateral stability, 716–717
  metabolic requirements, 723–724
  metal, 715–720
  for peripheral nerve injuries, 495
  pistoning, 722–723
  plastic, 720–723
  posterior stops, 715, 717–718, 722–723
  with prostheses, 90
  specialized designs for patient needs, 723–724
  Teufel, 723–725
  VAPC shoe clasp, 722–723, 725
Ankle/foot prostheses, 87, 127
Ankle fractures
  stress, 397
Ankle positioning
  for burn injuries, 611
Ankle sprains
  inversion, 397–399
  superficial peroneal neuropathy caused by, 541
  sural entrapment neuropathy caused by, 542
  tarsal tunnel syndrome caused by, 533
Ankylosis, 380–381
Anorexia
  after traumatic brain injury, 217
  and immobilization, 752
Anosmia, 283, 285
Anosognosia, 211
Antacids, 754
Anterior cord syndrome, 170–171
Anterior cruciate ligament (ACL), 392–393
Anterior drawer test, 392
Anterior interosseous nerve, 508
Anterior interosseous syndrome, 508–509
Anterior scalene syndrome, 501
Anterior stops
  on ankle-foot orthoses, 715–716, 718–720, 723
  on knee-ankle-foot orthoses, 729
Anterior tarsal tunnel, 538
Anterior tarsal tunnel syndrome, 538–540

- Antibiotics
  - for urinary tract infections, 585
  - *See also specific drug*
- Anticholinergic drugs
  - for traumatic brain injury, 212, 220
  - *See also specific drug*
- Anticholinesterases
  - for traumatic brain injury, 218
- Anticoagulants, 762–764
  - *See also specific drug*
- Anticonvulsants
  - for cranial neuropathies, 329, 335
  - for peripheral neuropathy, 477
  - for phantom limb pain, 146
  - for spinal cord injured patients, 191
  - for traumatic brain injury, 216, 221, 240
  - *See also specific drug*
- Antidepressants
  - *See* Tricyclic antidepressants; *specific drug*
- Antigravity positioning, 470–471, 604, 606–610, 628, 743, 746
- Antihypertensives
  - contraindications, 220
  - for spinal cord injured patients, 183–184
  - *See also specific drug*
- Antiinflammatory medications
  - for musculoskeletal injuries, 355, 357–358, 364, 372–373
  - *See also* Nonsteroidal antiinflammatory drugs (NSAIDs); *specific drug*
- Antiosteoclastic agents
  - for immobilization osteoporosis, 748, 750
- Antipersonnel mines
  - injuries caused by, 82
- Antispasticity agents
  - for traumatic brain injury, 216
  - *See also specific drug*
- Anxiety
  - and traumatic brain injury, 251–254
- Anxiolytics
  - and alcohol use, 240
  - for burn patients, 622
  - *See also specific drug*
- APDAB
  - *See* Army Physical Disability Appeals Board (APDAB)
- Aphonia, 332
- Appetite dysregulation
  - after traumatic brain injury, 217
  - and immobilization, 752
- Appraisal, 847
- Aquaplast, 617
- Archives of Physical Medicine and Rehabilitation, 4
- Arginine supplementation, 687
- Argyll Robertson pupil, 292, 297, 299
- Aris Isotoner, Inc., 665
- Arm amputations
  - *See* Upper extremity (UE) amputations
- Armed Forces Military Entrance Processing Stations
  - strength measurement methodology, 806
- Armed Service Amputation Center, 10, 149
- Arm scar compression, 673
- Arm supports
  - mobile, 676–677, 710–712
  - suspension sling, 712–713
- Army
  - aerobic capacity values, 804–806
  - basic recruit training, 814–816
  - body fat standards, 796–797
  - body weight/fat control program, 793
  - body weight limits, 796
  - cardiovascular risk screening program, 818
  - physical disability system, 865–883
  - physical therapy, 16, 20–28
  - rehabilitation teams, 16
- Army-Baylor University Graduate Program in Physical Therapy, 25
- Army Medical Command (MEDCOM), 866
- Army Medical Specialist Corps (AMSC), 23
- Army National Guard (ANG)
  - disability compensation, 882–883
- Army of the Republic of Vietnam (ARVN), 23
- Army Patient Administration Division (OTSG), 27
- Army Physical Disability Agency (USAPDA), 865–867
  - caseload statistics, 879–883
  - history, 865
  - location, 867
  - review by, 876–879
- Army Physical Disability Appeals Board (APDAB), 866
- Army Physical Therapy Registry (Persian Gulf War), 27–28
- Army Regulations 40-48 and 40-68, 24
- Army Reserve (USAR)
  - disability compensation system, 877, 882–883
- Arterial blood pressure
  - *See* Blood pressure (BP)
- Arthritis
  - in amputees, 142
- Arthrogenic contractures, 746
- Arthroscopy
  - peroneal neuropathies caused by, 529
- Artificial muscle
  - in hand orthoses, 708–709
- Ashworth scale
  - for grading muscle tone, 172
- ASIA
  - *See* American Spinal Injury Association (ASIA)
- Aspartate, 212
- Aspen Workgroup, 235
- Aspiration
  - in spinal cord injured patients, 182
- Assist mode, 599
- Ataxia
  - after traumatic brain injury, 217
- Atelectasis
  - in spinal cord injured patients, 182
- Atlanto-axial instability
  - C-1 to C-2, 174
- Atlanto-occipital dislocations, 173
- ATP
  - *See* Adenosine 5′-triphosphate (ATP)
- ATPase
  - *See* Adenosine triphosphatase (ATPase)
- Atrophy
  - optic, 287
  - *See also* Muscle atrophy
- Atropine, 183
- Attentional process disruption
  - after traumatic brain injury, 220
- Audiogram, 320
- Audiometry
  - pure tone, 319
- Auditory hallucinations, 319
- Auditory palpebral reflex, 313–314
- Auditory screening test, 319
- Auriculotemporal nerve syndrome, 303, 312
- Autonomic dysregulation
  - after traumatic brain injury, 217
- Autonomic hyperreflexia
  - in spinal cord injured patients, 183–184

Autonomic instability
  causalgia-associated, 482
Avellis's syndrome, 332
Axial flaps, 691
Axillary nerve, 496
  injury to, 499–500
Axillary straps
  for upper extremity prostheses, 57
Axon, 422–423
Axonal debris removal
  role of macrophages in, 433
Axonal degeneration
  See Wallerian degeneration
Axonal regeneration
  after peripheral nerve injury, 431–432, 434, 436
  after traumatic brain injury, 212
Axonal shear injuries, 209
Axonal transport, 425–426
  antegrade, 425
  models, 426
  retrograde, 425–426
Axon collateral sprouting
  after traumatic brain injury, 212–213
Axonotmesis, 429

B

Babinski response, 190
Bacitracin, 582, 591
Back pain
  immobilization for, 743
  low, 11, 378–389, 857
Baclofen
  for cranial neuropathies, 308
  for phantom limb pain, 146
  for spinal cord injuries, 191
  for traumatic brain injury, 216, 220
BAEPs
  See Brainstem auditory evoked potentials (BAEPs)
Balance
  with knee-ankle-foot orthoses, 730
Balance board training
  for musculoskeletal injuries, 398
Balkan Wars, 162
Ball-bearing mobile arm support, 710–712
Barbiturates, 216
Barium swallow, 327–328
Barthel Index-Granger Adaptation, 852
Barton, Walter E., 22
Barton Carey garments, 659, 669
Baruch, Bernard, 5
Baruch Committee, 5
Basic recruit training
  injuries associated with, 814–816
Bathing
  adaptive aids, 679
Batteries
  for myoelectric prostheses, 67
Battle fatigue
  prevention, 623
Battle stress, 16, 623
Battle wounds
  emotional reaction to, 846–848
BE amputations
  See Below elbow (BE) amputations
Becker-type ankle joint, 716
Bedrest
  See Immobilization; Immobility complications
Beds
  for spinal cord injured patients, 182, 186
  See also specific type of bed
Behavioral approaches
  to burn pain, 625
  to phantom limb pain, 147
Behavioral assessment tools, 223
Behavioral observations
  for neurobehavioral assessment, 242–243
Behavioral problems
  after traumatic brain injury, 211–213, 222–223, 238–239
Behrens, Charles F., 5
Belgian Army Medical Corps, 81
Bell clapping
  residual limb pain caused by, 142–143
Bell's palsy, 311
Below-elbow (BE) amputations, 43–44
  bilateral, 64
  long, 44, 52
  short, 44, 53
  surgical procedures, 46
  very short, 44, 53
Below-elbow prostheses, 34, 52–59
  versus above-elbow prostheses, 56
  choice of, 69–71
  elbow joints for, 55
  fitting, 48–50
  long, 44, 52
  myoelectric, 68–69
  and residual limb problems, 61
  short, 44, 53
  standard components, 55
  suspension and control, 54–56
  terminal devices, 44, 53–54
  very short, 44, 53
  wrist units, 53–55
Below-knee amputations (BKA), 82, 93–122
  classification, 93
  energy expenditure for walking, 93
  functional outcome, 83, 93
  high, 82
  indications, 94
  long, 93
  long-term follow-up, 106
  mortality rates, 93
  postoperative dressings, 96–100
  preoperative evaluation, 100–101
  psychological support, 101
  rehabilitation, 100–106, 143, 147–148
  residual limb lengthening, 96
  short, 93
  standard or medium length, 93
  surgical techniques, 93–96
  ultrashort, 93
  weight bearing, 98–99, 102–103
Below-knee prostheses, 106–120
  computer aided design/manufacturing, 120–121
  fitting, 106
  foot, 97–100, 110, 113
  immediate postoperative, 96–98
  selection, 106–107
  shanks, 112–113
  sockets, 106–110, 112
  suspension, 110–112
Bending moment
  at knee, 717, 723
Benedikt's syndrome, 296
Benserazide, 214–215

Benson's relaxation, 625–626
Benzodiazepines
  for burn patients, 622
  for traumatic brain injury, 216, 220–221, 240
  See also specific drug
Beta-blockers
  for causalgia, 490
  for phantom limb pain, 146
  for traumatic brain injury, 217, 220
  See also specific drug
Betadine
  See Povidone-iodine
Betapile, 668
Bethanecol, 218
Bevor's sign, 169
BIA
  See Brain Injury Association (BIA)
Bicycles
  stationary, 125, 635, 800–802
Biker's compression shorts, 657–658, 665
Billings, Frank, 4
Bine's test, 320
Biobrane, 582, 593, 629
Bio-Concepts, Inc., 665
Bioconcepts prefabricated glove, 667
Biofeedback
  for myoelectric prosthesis training, 69
  for phantom limb pain, 147
Biological dressings, 592–593
Bioplasty mesher, 590
Biphosphonates, 748, 750
Bivalved casts, 639, 747
Bladder calculi
  in spinal cord injured patients, 194
Bladder carcinoma
  in spinal cord injured patients, 194
Bladder drainage
  in immobilized patients, 585, 752
  in spinal cord injured patients, 193–194
Blink reflex, 302–303
  electrodiagnostic evaluation, 305
Blisters
  on burn wound, 632, 656
  foot, 818
  on residual limb, 136
Blocking, 463
Blood flow distribution, 791
Blood loss
  from debrided wound, 588
Blood pressure (BP)
  arterial, 790–791
  during exercise, 790–791
  in immobilized patients, 764–765
  response to maximal exercise, 791
Bobath shoulder roll, 713
Body composition
  definition, 792
  and physical capacity, 794–796
  and physical fitness, 792–799
  reference values, 792
  standards, 796–797
Body fat, 792–793
  android pattern, 793
  assessment, 793
  classification, 792
  essential or obligatory, 792
  excess, 793, 797
  gynoid pattern, 793
  measurement methodology, 797–799
  military control programs, 793, 796–799
  reduction, 793
  regional distribution, 793, 798
  storage, 792–793
Body fat percentage
  correlation between aerobic capacity and, 795
  and fitness level, 792–799
  military standards, 796–797
  reference values, 792
  values for military populations, 794
Body-powered prostheses
  hybrid (myoelectric and body-powered), 56, 59
  versus myoelectric prostheses, 68–71
  for upper extremity amputations, 53, 56, 62
Body strength
  See Strength
Body temperature
  regulation, 791
Body weight
  See Weight
Bolton, Frances P., 22
Bolton Bill, 22
Bone changes
  after burn injuries, 690
  in resistance training, 810
Bone homeostasis
  during immobilization, 747–748
Bone scans
  in causalgia, 487–488, 491
  in musculoskeletal injuries, 381, 385–386
  in reflex sympathetic dystrophy, 141–142
Bone spurs
  in residual limb, 139–140
Bone stress reactions
  See Stress fractures
Bonnier's syndrome, 303, 326
Boutonniere deformity, 611, 614, 639
Bowden control cable, 55, 57–58
Bowel programs
  for immobilized patients, 753
  for spinal cord injured patients, 185
Boyd hindfoot amputation, 86–87
Braces
  for musculoskeletal injuries, 374, 386
Brachial plexopathies, 496–498
  anatomic considerations, 496–497
  in burn patients, 689–690
  clinical presentation, 498–500
  electrodiagnosis, 498–500
  etiology, 497–498
Brachial plexus, 496–497
Brachial plexus injuries, 366
Bracing
  fracture cast, 735
  functional electrical stimulation for, 732–733
Bradyarrhythmia
  in spinal cord injured patients, 182–183
Bradycardia, 333
Brain
  contusions, 209
  surface anatomy, 223
Brain injuries
  See Traumatic brain injury (TBI)
Brain Injury Association (BIA), 227, 230
Brainstem auditory evoked potentials (BAEPs), 322–323
Brainstem tumors, 323
Brake mechanisms

  for knee prostheses, 127–128
Bretylium, 490
Bromocriptine, 214–215, 219–220, 222
Brown, Paul W., 10
Brown-Séquard syndrome, 170–171
Buf-N-Puff, 633
Bulimia
  after traumatic brain injury, 217
Bullet fragment removal
  from spinal cord injuries, 164–165
Bullets
  spinal cord injuries caused by, 164–165
  traumatic brain injury caused by, 208–209
Bullet shock waves
  spinal cord injuries caused by, 164
Bunnell, Sterling, 9, 35–36, 38
Bunnell splints, 709
Burn centers, 577, 583–584, 633
Burn claw deformity, 614
Burn cream, 582
Burn grafts
  See Skin grafts
Burn injuries, 575–702
  acid, 577–578
  acute phase, 595–625
  alkali, 577–578
  associated illnesses and injuries, 581
  chemical, 577–578, 583
  classification, 577–579
  closed treatment, 600
  complications, 582
  depth, 580–581
  dressings, 582, 587, 592–594, 600, 632
  electrical, 578–579, 591–592, 595, 691–692
  emergency care, 583–585
  first degree, 580
  flame, 577
  fourth degree, 581
  immobilization phase, 595–596, 625–631
  neurologic complications, 689–690
  nonhealing wounds, 587
  open treatment, 600
  Persian Gulf War, 14–15
  pigmentation, 605
  scald, 577
  second degree, 580–581
  severity factors, 579–581
  size, 579–580
  steam, 577
  surgical considerations, 582, 628
  thermal, 577
  third degree, 581
  topical treatment, 582
  treatment, 582–595
  wound excision, 587–588
  wound maturation phase, 595–596, 631–684
Burn Net, 600
Burn pain, 621–622
Burn patients
  adaptive equipment, 618–621, 631, 674–680
  airway management, 583–585, 598–599
  contracture prevention, 600, 603–610, 627–628, 636, 643–674
  edema management, 587, 600, 606, 655–674
  enteral feeding, 602, 687–688
  exercises, 596–597, 602–606, 627–628, 635–637, 652–653
  functional activities, 596, 601, 607, 618–621, 631, 674–680
  home management, 680–681
  inpatient care guidelines, 583–584
  intubation, 584, 598–600
  management, 595–693
  nutritional care, 602, 684–689
  orthotics, 596, 600, 611–618, 629–630, 637–643
  outpatient care, 583–584, 636
  pain management, 599–600, 621–623, 625–626
  phases of care, 595–596
  positioning, 607–611
  psychological support, 596, 623–626, 631, 633, 683–684
  pulmonary edema in, 584–585
  reconstructive surgery, 690–693
  rehabilitation goals, 595, 632
  return to duty, 637, 683–685
  vascular access, 584–585
Burn resuscitation formulas, 586
Burn scars
  assessment, 662–664, 666
  desensitization, 633–634
Burn shock
  fluid resuscitation for, 585–587
Burn splints, 615–616
Bursae injuries, 356
  residual limb pain caused by, 142
Bursitis
  trochanteric, 389
  in upper extremity amputees, 61
Burst fractures, 175
Bushnell General Hospital, Utah, 35, 80
Buspirone, 215–216
Bypass prosthesis, 126

C

Cable excursion
  in upper extremity prostheses, 59
Cable tensiometer, 802
Cacosmia, 283
CAD
  See Computer aided design (CAD)
CAI
  See Career Assessment Inventory (CAI) Enhanced Version
Calcaneal nerve, 533
Calcanectomy
  partial, 87
Calcarine cortex, 286, 289
Calcitonin
  for hypercalcemia, 750
  for immobilization osteoporosis, 748
  for phantom limb pain, 146, 148
Calcium alginate dressing, 594
Calcium channel blockers, 479, 490
Calcium gluconate, 578
Calcium intake
  during immobilization, 751
Calcium ions, 783–784
Calcium supplementation, 748
Callosities
  of residual limb, 137
Caloric testing, 321
Calorie counts
  for burn patients, 602, 687–688
  and immobilization, 752
CAM
  See Computer aided manufacturing (CAM)
Camouflage makeup
  for burn patients, 673, 690
Canada

  amputee vocational outcome study, 159
Canadian socket design, 132–133
Canes
  for above-knee amputees, 126
  for musculoskeletal injuries, 357
CANFIT system, 121
Canty, Thomas J., 10
Capsaicin, 477–478, 490
CAPTE
  See Commission on Accreditation for Physical Therapy Education (CAPTE)
Carafate
  See Sucralfate
Carbamazepine, 477
  for cranial neuropathies, 308, 335
  for traumatic brain injury, 216, 220–222
Carbidopa, 214–215, 219, 222
Carbohydrate metabolism
  and burn injuries, 687
Carbon Copy II, 92, 116, 120
Carbon Copy III, 116–117, 120
Carbon dioxide (CO_2)
  as orthotic power source, 705, 708–709
  partial pressure (PCO_2), 789, 791
  production, 788–789
Carbon fiber
  shoe shanks/inserts, 90
  socket reinforcement with, 91–92
Carbon graphite shoe inserts, 89–90
Carcinoma
  bladder, 194
  colon, 185
Cardiac enlargement
  in resistance training, 810
Cardiac output (C.O.)
  and blood flow distribution, 791
  maximum, 801
  and oxygen consumption, 790
  response to aerobic training, 809
  response to maximal exercise, 791
Cardiac reconditioning, 765
Cardiopulmonary resuscitation (CPR)
  after spinal cord injury, 162
Cardiorespiratory endurance
  See Aerobic power
Cardiovascular complications
  exertion-related, 818
  of immobilization, 762–765
  of spinal cord injury, 182–184
Cardiovascular conditioning
  and musculoskeletal injuries, 388
Cardiovascular deconditioning, 764–765
Cardiovascular function
  during exercise, 789–792
Cardiovascular response
  to aerobic training, 809
  to resistance training, 809–810
Cardiovascular risk screening program, 818
Career Assessment Inventory (CAI) Enhanced Version, 854
Career Compensation Act, 865, 879
Career content skills
  assessment, 855–856
Career plan guideline, 857
Career Planning Guide Book, 855
Caromed Int'l, Inc., 665
Carotid sinus reflex, 327, 333
Carpal tunnel, 509–510
Carpal tunnel release (CTR), 513–514
Carpal tunnel syndrome (CTS), 437–439, 509–514
  acute, 510, 513–514
  anatomic considerations, 509–510
  chronic, 510
  classification, 511–512
  clinical presentation, 511–512
  electrodiagnosis, 512–513
  etiology, 510–511
  treatment, 513–514
Casts
  bivalved, 639, 747
  fiberglass material, 48, 53, 97–100
  fracture bracing, 735
  plaster impression for residual limb, 121
  serial, 747
  thumb spica, 377
  See also Orthoses
CAT/CAM socket
  See Contoured Adducted Trochanteric/Controlled Alignment Method (CAT/CAM) socket
Catecholaminergic antagonists, 219–220
Catecholamines
  for traumatic brain injury, 212–215, 219
  See also specific drug
Catheters
  See Urinary catheters
Cauda equina, 168
  injuries, 191, 193, 732
Cauda equina syndrome, 170–172
Causalgia, 481–496
  cardinal characteristics, 482–483
  case studies, 491–496
  clinical considerations, 486
  clinical staging, 483
  diagnosis, 486–489, 491
  incidence, 481–482
  laboratory findings, 486–488
  pathophysiology, 483–485
  Persian Gulf War, 482, 491
  superficial radial nerve compression-associated, 505
  treatment, 489–491
  Vietnam War, 482
Cavernous sinus
  anatomy, 295
  inflammatory disease, 295
  septic thrombosis, 296
Cavernous sinus syndrome, 303
Cavitation, 747
CDP-choline
  for traumatic brain injury, 219
CED
  See Cognitive enhancing drug (CED)
Cellulitis
  in spinal cord injured patients, 187–188
Central Army Physical Review Council, 865
Central auditory pathway lesions, 318
Central cord syndrome, 170–171
Central nervous system (CNS)
  burn injury-associated damage, 633–634
  causalgia-associated changes in, 484
  effects of alcohol on, 240
  effects of immobility on, 749
  neural recovery, 212–213
Central venous pressure
  in immobilized patients, 764
Centripetal wrapping, 471
Centrophenoxine, 219
Cerebellar ataxia, 217

Cerebrospinal fluid markers
  in traumatic brain injury, 211
Cerebrovascular blood flow
  and cognitive function, 219
Cerium nitrate, 591
Cervical collars, 361
Cervical glides
  dorsal, 362
Cervical radiculopathy, 363–366
Cervical soft tissue injuries, 361–363
  rehabilitation, 361–362
Cervical spine, 167–168
Cervical spine injuries, 169, 171, 173–174
  complications, 181–182, 191, 193
  interdisciplinary team management case study, 843–844
Cervical spine orthoses, 174
Cervical stabilization exercises, 364–365
Cervical stenosis
  congenital, 165, 171
Cervical traction, 364
Cervicothoracic disorders, 361–367
  See also specific disorder
Cervicothoracic stabilization exercises, 364–365
Chance fractures, 175
Charcot's spine, 189
Check sockets, 52–53, 121, 130
Chemical burns, 577–578
  treatment, 583
Chemosensory testing, 285
Chest expansion
  elbow lock operation by, 59–60
Chest strap
  for above-elbow prostheses, 58
  for humeral neck amputation prostheses, 60
Chief of Army Medical Specialist Corps Clinical Investigation and Research (AMSC-CIR), 27
Childbirth
  in spinal cord injured patients, 195
Children
  skin graft sites, 595
Chin control
  of wheelchair, 177
Chlorhexidine, 597
Chlorphenesin, 308
Choking
  socket, 143
Cholelithiasis
  in spinal cord injured patients, 185
Choline, 218
Cholinergic substances
  for traumatic brain injury, 216, 218–220
  See also specific drug
Chopart's amputations
  See Tarsotarsal amputations
Chorda tympani syndrome, 312
Chronaxie, 449
Ciliary muscle
  innervation, 292–293
Cimetidine, 754
Circulation, motion, sensation, and temperature (CMST), 616, 626
Circulatory complications
  after amputation, 137–139
  in immobilized patients, 764–765
Citalopram, 216
Civil War
  amputations, 34, 71–72, 80, 134
  causalgia, 481
  cranial neuropathies, 339
  immobility complications, 742
CK
  See Creatine kinase (CK)
Claymore mines
  injuries caused by, 82
Clear film dressings, 594–595
Cleveland Clinic, 5
Clinical symptom complex, 356
Clinitron bed, 186–187, 628, 631, 757
Clodronate, 748, 750
Clonazepam, 220–222, 477
Clonidine, 219–220, 479, 490
CMAPs
  See Compound motor action potentials (CMAPs)
CMRR
  See Common mode rejection ratio (CMRR)
CMST
  See Circulation, motion, sensation, and temperature (CMST)
CN
  See Cranial nerves (CN)
CNS
  See Central nervous system (CNS)
C.O.
  See Cardiac output (C.O.)
Coagulation necrosis
  caused by acid burns, 577–578
Coban, 471, 613, 658–659
Cochlear implants, 324
Cochlear nerve
  anatomy and function, 315–317
Cochlear neuropathies, 318–324
  evaluation, 319–322
  management, 323–324
Cocked-hat flap procedure, 37
Cockup splint, 709–710
Codeine, 622
Code of Hammurabi, 864
Cognitive-behavioral dysfunction, 217–220, 222–223, 230
  and family outcome, 252–253
  treatment, 218–219, 228, 237–246
Cognitive-behavioral profiling
  after traumatic brain injury, 217
Cognitive enhancing drug (CED), 218
Cognitive function
  drugs that may enhance, 218–220
  drugs that may interfere with, 219–220
Cognitive remediation
  for brain injured patients, 245
Cohen, Henry, 429
Colace
  See Docusate sodium
Cold exposure
  differential sensitivity of nerves to, 445
  effect on nerve conduction velocity, 445
  and exercise-related injuries, 819–820
  nerve damage caused by, 443–446
  tissue injury from, 123
  See also Frostbite
Cold-induced neuropathies, 443–446
  clinical considerations, 443–444
  and duration of exposure, 445
  electrodiagnosis, 445–446
  morphologic changes, 445
  pathophysiology, 444–445
  prognosis, 444
  and temperature considerations, 445

  and type of nerve involved, 445
Collagen
  and hypertrophic scar formation, 644
Collagen biosynthesis
  in denervated muscles, 435, 465
Collagen production phase, 354–355
Colle's fracture
  carpal tunnel syndrome caused by, 510, 513
Collet-Sicard syndrome, 331–332, 336, 340
Colon carcinoma
  in spinal cord injured patients, 185
Colorectal disease
  in spinal cord injured patients, 185
Columns of Denis, 174–175
Coma
  definition, 234
Coma/Near Coma Scale, 224
Coma Recovery Scale, 224
Coma stimulation programs, 235–237
Comatose patient
  cranial neuropathies in, 280
Combat boots
  as risk factor for injury, 817
Combat stress, 16, 623
Combat theater
  physical medicine and rehabilitation in, 8–9
  physical therapy in, 19–30
  See also specific theater
Commission on Accreditation for Physical Therapy Education (CAPTE), 25
Common mode rejection ratio (CMRR), 452
Communication
  adaptive devices, 620–621, 681–682, 833
  by intubated patients, 181
Community mobility
  and spinal cord injured patients, 178–179
Community reintegration, 845–861
  for spinal cord injured patients, 180
  for traumatic brain injured patients, 224, 227–229
  See also Return to duty; Vocational rehabilitation
Compartment syndromes, 439–440
  pronator teres syndrome associated with, 506
Compensation Act, 865, 879
Compensation systems
  See Physical disability system
Complex regional pain syndrome (CRPS)
  diagnostic criteria, 486–489
  See also Causalgia; Reflex sympathetic dystrophy (RSD)
Complex repetitive discharges, 455
Compound motor action potentials (CMAPs)
  amplitude, 459, 464
  inability to elicit, 460
  measurement, 451
  in ulnar neuropathy, 462, 517
Compression
  for edema control, 471
  for musculoskeletal disorders, 357
Compression inserts
  for orthoses, 650, 664–674
Compression neuropathy, 436–439
  classification, 429
  clinical considerations, 437–439
  and impulse propagation, 450
  pathophysiology, 436–437
  See also specific nerve
Computed tomography (CT)
  in cranial neuropathies, 285, 299, 341
  in femoral neuropathy, 527
  in musculoskeletal disorders, 381
  socket design with, 121
  in spinal cord injury, 163–164, 173, 175
  in traumatic brain injury, 225, 230
Computer aided design (CAD)
  of sockets, 120–121
Computer aided manufacturing (CAM)
  of sockets, 120–121
Computer aided socket design (CASD), 121
Computers
  for spinal cord injured patients, 179
Concentric contractions, 783
Concentric needle electrodes, 453
  bipolar, 453
  coaxial, 453
Concussive injuries, 227, 230
  classification, 230
  incidence, 210
  symptoms, 227, 230–231
Conditioning period
  and muscle injury avoidance, 356
Condom catheters
  skin complications, 186–187
Conduction block, 449–450
  focal, 430
Conduction slowing with temporal dispersion
  electrodiagnostic findings, 461–462
Conduction velocity
  See Nerve conduction velocity
Cones, 285–286
Confusion
  in burn patients, 631
Connecticut Chemosensory Clinical Research Center, 283–284
Connective tissue
  denervation-associated changes, 435, 438–439, 442
  of nerve, 426–427
  peripheral neuropathy-associated stretching, 465–467
Consciousness
  loss of, 230, 234–235
Constipation
  in immobilized patients, 752–753
Contact dermatitis
  caused by orthoses, 668–669
  on residual limb, 138–139
Continental United States (CONUS) hospitals
  physical medicine and rehabilitation at, 9
  See also specific hospital
Continuous passive motion (CPM) devices, 605, 616–618, 639–641, 747
Continuous positive airway pressure (CPAP), 599
Contoured Adducted Trochanteric/Controlled Alignment Method (CAT/CAM) socket, 129–130
Contour foam, 670
Contracture prevention, 8–9, 226, 746–747
  in burn patients, 600, 603–610, 627–628, 636, 643–674
  in lower extremity amputees, 81, 84, 88, 94, 101–102, 124–125, 135
  in spinal cord injured patients, 189
  in upper extremity amputees, 47
Contractures
  arthrogenic, 746
  formation, 745
  during immobilization, 745–746
  myogenic, 435, 465–467, 491, 746
  soft tissue, 746
  types, 746
Control mode, 599
Control systems

  above-elbow prostheses, 57–58
  below-elbow prostheses, 54–56
  humeral neck amputation prostheses, 60
  myoelectric prostheses, 65–67
Contusions
  brain, 209
  lumbar, 386–389
CONUS
  See Continental United States (CONUS) hospitals
Conus medullaris, 168
  injuries, 193
Conus medullaris syndrome, 170, 172
Convalescent hospitals
  pre-World War II, 5
Convalescent leave, 872
Convergence system, 294
Coping skills
  and vocational rehabilitation, 846
Cordectomy, 192
Corneal reflex, 302–303
  electrodiagnostic evaluation, 306–307
Coronary artery disease
  in spinal cord injured patients, 184
Corps of Invalids, 72
Cortical blindness, 287, 291
Corticosteroids
  for causalgia, 490
  for musculoskeletal injuries, 358, 361, 366–367, 374, 377
  See also specific drug
Corticotropin-releasing hormone, 217
Cortisone, 542, 544
Cosmetic covers
  for above-knee prostheses, 132
  for foot prostheses, 117, 119
  for myoelectric prostheses, 68–69, 71
Cosmetic prostheses
  hand, 43
Costoclavicular syndrome, 501
Cough, 333
  impaired, 182
Coulter, John S., 5
Coumadin, 183
Council on Medical Education, 5
Council on Physical Therapy (American Medical Association), 22
Counseling
  See Emotional counseling; Psychological support; Psychotherapy
Counterforce braces
  for epicondylitis, 374
Courage
  as characteristic of soldier, 846
CP
  See Creatine phosphate (CP)
CPAP
  See Continuous positive airway pressure (CPAP)
CPM devices
  See Continuous passive motion (CPM) devices
C-polymodal nociceptors (C-PMN), 484–485
CPR
  See Cardiopulmonary resuscitation (CPR)
Cramp
  heat, 819
Cranial nerves (CN), 280
  anatomy and function, 280–281
  decompression, 280
  I. *See* Olfactory nerve
  II. *See* Optic nerve
  III. *See* Oculomotor nerve
  IV. *See* Trochlear nerve
  V. *See* Trigeminal nerve
  V_1. *See* Ophthalmic nerve
  V_2. *See* Maxillary nerve
  V_3. *See* Mandibular nerve
  VI. *See* Abducens nerve
  VII. *See* Facial nerve
  VIII. *See* Acoustic nerve
  IX. *See* Glossopharyngeal nerve
  X. *See* Vagus nerve
  XI. *See* Accessory nerve
  XII. *See* Hypoglossal nerve
Cranial neuropathies, 279–352
  causes, 280
  in comatose patient, 280
  incidence, 280
  prognosis, 280
  See also specific cranial nerve
Creatine kinase (CK), 579
Creatine phosphate (CP), 786–787
Cricopharyngeus sphincter dilation, 329
Cricothyroid muscle, 333
Croatia, 28
Crocodile tears, 312
Cross-bridges, 783–784
Crossed adduction maneuver, 371
CRPS
  See Complex regional pain syndrome (CRPS)
Crush injury
  carpal tunnel syndrome caused by, 510, 514
Crutch ambulation
  for above-knee amputees, 126
  carpal tunnel syndrome caused by, 510–511
  by multiple amputees, 148
  for musculoskeletal injuries, 357
  patient education, 102–104, 106
  for spinal cord injured patients, 178
Crutch palsy, 503–504
Cryotherapy
  for musculoskeletal injuries, 357–358, 371–373, 383
CT
  See Computed tomography (CT)
CTR
  See Carpal tunnel release (CTR)
CTS
  See Carpal tunnel syndrome (CTS)
Cubital tunnel, 514–515
Cubital tunnel release, 519
Cubital tunnel syndrome, 514, 516, 519
Cultural values
  and counseling approaches, 849
Cultured autologous kertinocite grafts, 591–592
Cutaneous sensation
  and neurological evaluation of spinal cord injury, 169
Cuthbertson, D. P., 742
Cybex dynamometer, 635, 801
Cyclanedelate, 219
Cycle ergometry, 800, 802
Cycling tests
  for anaerobic power, 801–802
Cyproheptadine, 217
Cysts
  epidermoid, 137
Cytidene 5′ -diphosphocholine
  See CDP-choline
Cytotec
  See Misoprostol

D

Daily living skills
  See Activities of daily living (ADL)
Dantrolene sodium, 217
Deafferentation pain, 475
Death
  accidental, 209–210
  exertion-related, 817
Debridement
  burn wounds, 587–589, 597
  of devitalized tissues, 81, 135
  enzymatic, 588–589
  instruments, 587–588
  for pressure sores, 187
  See also Excision
Decompression sickness, 166
Deconditioning
  cardiovascular, 764–765
  prevention, 8–9, 81, 84, 126
Deep brain stimulation
  for phantom limb pain, 146
Deep heating, 467, 746–747
Deep peroneal nerve, 538
Deep venous thrombosis (DVT), 183, 762–764
  differential diagnosis, 763–764
  prevention, 763–764
  versus pulmonary embolism, 763
Degenerative arthritis
  in amputees, 142
Degloving injuries, 37
Dehydration, 819
Dejerine's anterior bulbar syndrome, 340
Deltoid aid, 639
Dematomes
  for spinal cord injury evaluation, 168
Dementia
  immobility-related, 749
Demerol, 495
Demyelination
  See Conduction block
Dendrites, 422–423
Denervation
  end organ changes following, 431–436
  muscle changes following, 434–436
Denervation supersensitivity, 435–436, 456
  after traumatic brain injury, 213
Dens fractures, 174
Dental anesthesia
  ocular complications, 296
Dentistry
  and oral contractures, 638
  role in medical evaluation board, 868
Department of Defense (DoD)
  disability directives, 865
Department of Veterans Affairs
  See Veterans Administration
L-deprenyl
  for traumatic brain injury, 214
Depression
  after traumatic brain injury, 242, 245–246, 251–254
  in spinal cord injured patients, 196
De Quervain's tenosynovitis, 376–377
Dermapad, 670
Dermatitis
  contact, 138–139, 668–669
  stasis, 137–138
Dermis
  artificial, 592
Desensitization
  of burn scar, 633–634
  hyperesthetic, 472–473, 491–492, 495
Desipramine, 219, 476
Detraining, 808–809
Detrusor-sphinctor dyssynergia
  in spinal cord injured patients, 193–194
DEXA
  See Dual photon x-ray absorptiometry (DEXA)
Dexamethasone, 315, 377
Dextroamphetamine, 214–215
Diabetes mellitus
  in spinal cord injured patients, 185
Diabetic ischemic neuropathy, 296
Diarrhea, 333
Diathermy, 467, 746–747
  microwave, 746
  ultrasound, 467, 746–747, 758
Diazepam, 213
Didronel, 221
Dietary considerations
  See Nutrition
Dietary fiber
  for immobilized patients, 753
Dietitians, 22–23
Dietrick, J. E., 742
Differential amplification
  electrodiagnostic, 451–452
Digital control systems
  for myoelectric prostheses, 65
Digital neuropathies, 543–545
  anatomic considerations, 543
  clinical presentation, 544
  differential diagnosis, 544
  electrodiagnosis, 544
  etiology, 543–544
  treatment, 544–545
Digits
  See Fingers
Dihydroergotamine, 219
Diltiazem, 479
DIP disarticulation
  See Distal interphalangeal (DIP) disarticulation
Diphosphonate EHDP, 748
Diplopia, 297
  management, 299–300
Disability event
  adaptive stages to, 847–848
Disability ratings, 873–875
Disability Rating Scale, 224
Disarticulation
  and residual limb/prosthesis interface, 89
  See also specific type of disarticulation
Discography, 381
Disks
  herniations, 381
  internally disrupted, 380
Disk space
  narrowing, 380
Dislocations
  anterior interosseous syndrome caused by, 508–509
  brachial plexopathies caused by, 497, 499
  neuropathies associated with, 440–443
  pronator teres syndrome caused by, 506–507
Disodium diphosphonate, 748
Disodium ethane-1-hydroxy-1,1-diphosphonate, 748
Distal interphalangeal (DIP) disarticulation, 37

Distal metatarsal amputations, 85
Division of Special Hospitals and Physical Reconstruction, 830–831
Dizziness, 319
Docusate sodium, 753
DoD
  See Department of Defense (DoD)
Dopamine, 219
Dopamine agonists
  for traumatic brain injury, 213–215, 217, 220–222
  See also specific drug
Doppler ultrasonography
  color-flow, 763
Dorrance hooks, 53
Dorsal cord stimulation
  for phantom limb pain, 146–148
Dorsal root entry zone (DREZ) lesioning, 146, 148, 192
Dorsal root ganglia, 167–168
Dorsal scapular nerve, 496
Dorsiflexion assist orthosis
  spring wire, 715
Dorsiflexion (anterior) stops
  on ankle-foot orthoses, 715–716, 718–720, 723
  on knee-ankle-foot orthoses, 729
Double-stopped ankle joint, 716
Dow Corning prosthetic foam, 670
Dow Corning silicone gel sheeting, 669–670
Downey Hand Center, 472–473
Doxepin, 476–477, 492
Dressing
  adaptive aids, 679–680
  See also Activities of daily living (ADL)
Dressings
  biological, 592–593
  for burn wounds, 582, 587, 592–594, 600, 632
  calcium alginate, 594
  clear film, 594–595
  hydrocolloid, 594
  local compression, 357
  for lower extremity amputations, 84, 87–88, 94, 96–100, 102–106, 124–125
  postgraft changing, 626–627
  for pressure sores, 187
  rigid, 87–88, 96, 99
  for skin grafts, 591–594, 626–627, 629
  soft, 96, 125
  synthetic, 593–594
  for upper extremity amputations, 48–50
  See also Immediate postoperative rigid dressing (IPORD); *specific type of dressing*
DREZ lesioning
  See Dorsal root entry zone (DREZ) lesioning
Drinking
  adaptive devices, 619–620
Driving
  adaptive aids, 54, 64, 178–179, 682
  and brain injured patients, 227
Dual-control cable
  for upper extremity prostheses, 57–60
Dual photon x-ray absorptiometry (DEXA), 799
Duane's retraction syndrome, 296
Duoderm, 594, 656, 662, 673
Durnin-Womersley equations, 797–798
DVT
  See Deep venous thrombosis (DVT)
Dycem, 620
Dynamic constant resistance
  See Isotonic exercises
Dynamic response feet, 92, 115–120, 127
Dynamic splinting, 747
Dynamic strength
  measurement, 802
Dynamometer, 802
  Cybex, 635, 801
  isokinetic, 802–803
Dysarthria, 312, 340–341
Dysautonomia
  central, 211
Dysesthetic pain, 475
Dysgeusia, 311
Dyskinesias
  after traumatic brain injury, 221
Dysosmia, 283, 285
Dysphagia, 332, 334–335
  after traumatic brain injury, 222
Dysphonia, 332
Dysreflexia
  autonomic, 183–184
Dystonia
  after traumatic brain injury, 220
Dysvascular amputees
  bilateral, 149
  residual limb pain, 139

E

Ear
  anatomy, 315–317
  burn injury positioning concerns, 608
  prosthetic, 655
Early recovery management programs (ERMPs)
  for brain injured patients, 226, 236
Eccentric contractions, 783, 813
Ectopic bone
  in residual limb, 139–140
ECU
  See Environmental control unit (ECU)
Edema
  after peripheral neuropathy, 470–471, 491–492, 496
  in burn patients, 587, 600, 606, 655–674
  physiology, 470
  prevention, 88, 104–106, 124–125
  pulmonary, 584–585, 820
  stump, 134, 138
Edema-sleeves, 658
Edinger-Westphal nucleus, 286, 291–292
Education
  See Patient education; Family education
Educational experiences
  and vocational assessment, 851
EEG
  See Electroencephalography (EEG)
Efferent fibers
  in cranial nerves, 281
Elastic abdominal binders
  for spinal cord injured patient, 182, 184
Elastic bands
  for below-elbow prostheses, 55
Elastic digi-sleeves, 658
Elastic hoods, 650
Elastic sport garments, 665
Elastic stockinettes
  for amputations, 48–50, 94, 97, 125
Elastic stretch, 465
Elastic tubular supports, 658–660
Elastic wrapping

  for burn patients, 601, 606, 610–611, 613, 615, 630, 645, 656, 658–660
  for lower extremity amputations, 87–88, 94, 96–98, 103–105, 124–125
  for upper extremity amputations, 48–50
Elastin, 355
Elastogel, 667, 669–670
Elastomers
  in orthotic inserts and overlays, 670
Elastomer silicone shoe inserts, 642–643
Elbow
  contracture prevention, 47
  cubital tunnel at, 514–516, 519
  immobilization orthoses, 630
  positioning for burn injuries, 610
  scar compression, 672–673
  ulnar nerve injury at, 514–519
Elbow disarticulation, 44
  prostheses, 44
  surgical procedures, 46
Elbow disorders, 372–376
  rehabilitation, 376
Elbow flexion test, 515
Elbow-locking cable
  for upper extremity prostheses, 57–60
Elbow orthoses, 639
  functional, 713
  static, 713
  support of, 713–715
Elderly
  skin graft sites, 595
Electrical arc, 578
Electrical burns, 578–579
  combined with thermal injuries, 578–579
  treatment, 591–592, 595, 691–692
Electrical motor
  as orthotic power source, 705, 708–709
Electrical muscle stimulation
  for peripheral nerve injuries, 481
Electrical stimulation
  for cranial neuropathies, 315
  for edema control, 470–471
  for musculoskeletal injuries, 358–359, 374, 383
  for peripheral nerve injuries, 479–481
  for spinal cord injured patients, 192
  See also Transcutaneous electrical nerve stimulation (TENS)
Electrical stimulators
  implantable functional, 179
Electrocochleography, 322
Electrodes
  electrodiagnostic, 453
  electroencephalographi, 284–285
  electromyographic, 453–454
  ground, 453
  myoelectric, 65, 67
  needle, 453
  surface, 453, 456–457
  transcutaneous electrical nerve stimulation, 480
Electrodiagnosis, 11–12
  of accessory neuropathies, 338
  of acoustic neuropathies, 322–323
  amplification, 451–452
  of anterior interosseous syndrome, 509
  of anterior tarsal tunnel syndrome, 539
  of brachial plexopathies, 498–500
  of carpal tunnel syndrome, 512–513
  classes of disorders detectable by, 448
  of cold-induced nerve injuries, 445–446
  in compartmental syndromes, 440
  and determination of injury severity, 459–462
  of digital neuropathies, 544
  electrodes, 453
  of facial neuropathies, 314–315
  of femoral neuropathy, 526–527
  filters, 452–453
  of foot and ankle nerve injuries, 531
  general principles, 448
  of glossopharyngeal neuropathies, 328
  history, 420
  of hypoglossal neuropathies, 340–341
  instrumentation, 450–453
  of ischemic nerve injuries, 440
  limitations, 464–465
  localization with, 461–463
  of lumbosacral plexopathy, 523–524
  of oculomotor neuropathies, 299
  of olfactory neuropathies, 284–285
  of peripheral nerve injuries, 448–465
  of peroneal neuropathy, 529–530
  of posterior interosseous nerve entrapment, 505
  of pronator teres syndrome, 508
  of radial neuropathy, 503
  recovery monitoring with, 460, 463–464, 468
  of residual limb pain, 142
  of sciatic neuropathy, 525–526
  of superficial peroneal neuropathy, 541
  of supracondylar process syndrome, 506
  of sural entrapment neuropathy, 543
  of tarsal tunnel syndrome, 535
  of thoracic outlet syndrome, 501–502
  timing of, 460–461
  of trigeminal neuropathies, 305
  of ulnar neuropathy, 514, 517–519, 521–522
  utility of, 459–465
  of vagus neuropathies, 333–334
  See also specific instrument
Electroencephalography (EEG)
  in olfactory nerve injuries, 284–285
Electrogustometry, 314
Electrolyte homeostasis, 750
Electromechanical digitization
  for socket design, 121, 130
Electromyography (EMG), 453–458
  in anterior interosseous syndrome, 509
  in brachial plexopathies, 499
  in carpal tunnel syndrome, 512–513
  and determination of injury severity, 459–462
  electrodes, 453–454
  in femoral neuropathy, 527
  filter settings, 452
  in foot and ankle nerve injuries, 531
  in hypoglossal neuropathies, 340–341
  insertional activity, 454
  instrumentation, 450–453
  of laryngeal nerves, 330, 333–334
  limitations, 464–465
  in lumbosacral plexopathy, 523
  at maximal contraction, 458
  at minimal contraction, 456
  motor unit electrical morphology, 456–457
  motor unit recruitment, 457–458
  in peroneal neuropathy, 530
  in posterior interosseous nerve entrapment, 505
  in pronator teres syndrome, 508
  recovery monitoring, 463–464

    at rest, 454–456
    in sciatic neuropathy, 525
    in supracondylar process syndrome, 506
    in ulnar neuropathy, 519
    See also Nerve conduction studies (NCS)
Electronystagmography (ENG), 322
Electrophysiologic assessment
    of spinal cord injury, 173
    of traumatic brain injury, 211
Electroretinography (ERG), 290
Electrotherapy, 830
Elevation
    See Antigravity positioning
El Salvador, 28
Elset wrap, 658–659
Emergency medical care
    burn injuries, 583–585
    spinal cord injury, 162
    traumatic brain injury, 225
EMG
    See Electromyography (EMG)
Emotional counseling
    for brain injured patients, 227–228
    for burn patients, 596, 623–626, 631, 633, 683–684
    cultural concerns, 849
    group, 849
    for hearing-impaired patients, 325
    for immobilized patients, 750
    in interdisciplinary team management, 832
    for lower extremity amputees, 84, 88, 101, 125, 133–134
    resistance to, 850
    for spinal cord injured patients, 180, 195–196
    for upper extremity amputees, 47, 52
    in vocational rehabilitation, 848–850
    See also Psychological support; Psychotherapy
Emotional needs
    following injury, 847
Emotional reaction
    to war-related injuries, 846–848
Emotional sequelae
    of traumatic brain injury, 238–239, 243–246, 253
Endocrine adaptation
    in resistance training, 810
Endocrinologic complications
    of immobility, 750–751
    of spinal cord injury, 185
Endoneurium, 427
    transection, 430–431
End organ changes
    following denervation, 431–436
Endurance
    See Aerobic power; Anaerobic power
Energy expenditure for walking
    with ankle-foot orthoses, 723–724
    at different levels of lower extremity amputations, 92–93
    with functional electrical stimulation, 733
    of hemiplegic ambulation, 723
    with knee-ankle-foot orthoses, 729
    measurement, 723
    by multiple amputees, 149
    of normal ambulation, 724
    of paraplegic ambulation, 731
    with reciprocating gait orthoses, 732
    by spinal cord injured patients, 178
    of wheelchair locomotion, 731
Energy needs
    of burn patients, 684–686
Energy transfer
    in exercise, 787–788
Energy transformation
    within muscle, 786
ENG
    See Electronystagmography (ENG)
Enteral feeding
    of burn patients, 602, 687–688
Entrapment syndromes
    of foot and ankle, 531–545
    lower extremity, 522–531
    upper extremity, 496–522
    See also Compression neuropathy; *specific nerve*
Environmental control systems
    adaptive devices, 621
Environmental control unit (ECU)
    for spinal cord injured patients, 177, 179
Environmental stimulation
    and comatose patient, 236
Enzymatic debridement, 588–589
Ephedrine sulfate, 184
Epicondylar muscles
    stretching, 373
Epicondylitis, 355, 372
    differential diagnosis, 372
    lateral, 372–374
    medial, 372–374
    rehabilitation, 373–374
Epidermoid cysts
    on residual limb, 137
Epidural anesthesia
    for lumbar pain, 382
    for residual limb pain, 142, 147
Epilepsy
    posttraumatic, 221
Epineurium, 427
    transection, 431
Épluchage, 597
Equinus deformities, 83
ERG
    See Electroretinography (ERG)
Ergometry
    cycle, 800, 802
Ergot alkaloids
    for traumatic brain injury, 215, 219
    See also specific drug
ERMPs
    See Early recovery management programs (ERMPs)
ERTL procedure
    for lower extremity amputation, 95–96
Esophageal dysfunction, 333
Esophageal manometry, 334
Ethacrynic acid, 750
Ethical issues
    with traumatic brain injury, 235
Ethnic groups
    counseling concerns, 849
Etidronate disodium, 221, 748, 750
Etiracetam, 219
Evoked potentials
    gustatory, 314
    olfactory, 284
    trigeminal, 307–308
    visual, 290–291
Excision, 587–588, 597
    to fascia, 588
    instruments, 587–588
    tangential, 587–588
Exercise(s)

  acute responses to, 791
  for burn patients, 596–597, 602–606, 627–628, 635–637, 652–653
  cardiovascular function during, 789–792
  energy generating systems for, 782
  energy transfer in, 787–788
  for immobility complications, 744, 765
  for lower extremity amputees, 88, 102–103, 123, 125
  medical problems associated with, 814–820
  mental concentration during, 635
  for musculoskeletal injuries, 355, 358, 383–385, 387–389
  for peripheral neuropathy, 469–470, 492, 495–496
  physiological principles, 787–792
  pulmonary function during, 788–789, 801
  for spinal cord injury, 167
  See also Physical capacity for exercise; Physical training; *specific type of exercise*
Exercise intolerance
  in immobilized patients, 765
Exertional rhabdomyolysis, 818
Exidine
  See Chlorhexidine
Exophthalmus, 297
Exoskeleton prostheses, 132
Expectancy
  principle of, 623
Explosions
  burns caused by, 581
Extension exercises
  for musculoskeletal injuries, 383
Extensor spasms
  in spinal cord injured patients, 190–191
Exteroceptive sensations
  phantom limb, 143
Eye closure
  reflex, 313–314
Eyelid muscles
  innervation, 294
Eye movement
  clinical evaluation, 299
  muscular control, 293–294
  supranuclear control system, 294
Ezeform, 617

F

Facial dyskinesis, 312
Facial exercise program
  for burn patients, 653
Facial movement
  innervation, 310–311
  testing, 312–313
Facial myokymia, 314–315
Facial nerve, 308–315
  anatomy and function, 308–311
Facial neuropathies, 311–315
  electrodiagnosis, 314–315
  evaluation, 312–314
  management, 315
  prognosis, 315
  symptoms and signs, 312
  syndromes, 311–312
Facial orthoses
  compression inserts, 650
  fitting, 652–655
  immobilization, 630
  transparent, 638, 650–655
  wearing instructions, 651–652, 655
Facial pain, 304, 308
Facial paralysis, 311–312
Facial scars
  camouflage makeup, 673, 690
  management, 638
  plastic surgery strategies, 692–693
FAD
  See Flavin adenine dinucleotide (FAD)
  See McMaster Family Assessment Devise (FAD)
Fading
  of phantom limb sensation, 143
Falklands War, 443
Family
  role in interdisciplinary team, 833
Family Adaptability and Cohesion Scales, 254
Family assessment
  in vocational rehabilitation, 853–854
Family education
  after brain injuries, 227–228, 231, 245, 254
  after burn injury, 601–602, 624
  after spinal cord injury, 180
Family Environment Scale, 254
Family Needs Questionnaire (FNQ), 254
Family outcome
  after traumatic brain injury, 250–255
Famotidine, 754
Fascia
  excision to, 588
  graft adherence on, 588
Fascicular composition
  of peripheral nerves, 426–427, 435, 438–439, 442
Fasciculation potentials, 455
Fasciitis, 355
  plantar, 400–401
Fast twitch (type 2) fibers, 784, 802, 809
Fat
  See Body fat
Fat-free mass, 792
  correlation between body strength and, 795–796
Fatigue
  muscle, 786
Fat intake
  and burn injuries, 687
FDA
  See Food and Drug Administration (FDA)
Fecal impaction
  in immobilized patients, 753
  in spinal cord injured patients, 185
Feeding
  adaptive devices, 619–620, 676–677
Felbamate, 222
Femoral neck stress fractures, 397
Femoral nerve, 526
Femoral neuropathy, 526–527
  anatomic considerations, 526
  clinical presentation, 527
  electrodiagnosis, 526–527
  etiology, 526–527
  treatment, 527
Fenfluramine, 217
Fentanyl (Fentaugh), 622
Fertility
  in spinal cord injured patients, 194–195
FES
  See Functional electrical stimulation (FES)
Fiberglass cast material, 48, 53, 97–100
Fiberglass orthoses, 617
Fibrillation potentials, 454, 494

  quantification, 455
Fibrillations, 454
Fibroblasts
  and hypertrophic scar formation, 644
Fibula
  excess length, 140
  hypermobile, 140
  stress fractures, 397
Fifth ray amputations, 86
Figure-8 clavicle strap, 638–639
Figure-8 elastic bandage wraps, 656
Figure-8 harness, 55–58
Figure-8 wrapping technique, 88, 104–105, 125
Filters
  electrodiagnostic, 452–453
Finances
  and brain injured patients, 229
  and spinal cord injured patients, 180
Finger extension attachments
  for hand orthoses, 705–706
Finger prostheses, 39
  extension, 39
  fabrication, 39
  See also Thumb prostheses
Fingers
  lengthening procedures, 36
  pollicization, 36–37
  scar compression, 671
  single amputation, 37
  See also Digital neuropathies
Finger splint, 615
Finger wraps, 613
Finkelstein's test, 376
Finnish War, 15
First Conference of the International Federation of Automatic Control, 65
Fish-mouth flaps
  for transfemoral amputation, 124
Fitness
  See Aerobic power; Physical fitness
Fitzsimons Army Hospital, Colorado, 10, 52, 80, 148–149
Fixation
  for limb length preservation, 95
Fixed ankle orthoses, 734–735
Flame burns, 577
Flavin adenine dinucleotide (FAD), 786
Flex Foot, 116–117, 120
  variants, 117–119
Flexibility
  decreased, 358–359
  as risk factor for injury, 817
Flexibility exercises
  for musculoskeletal injuries, 356, 359, 367–369, 386–387, 395
Flexicare bed, 757
Flexion exercises
  for hamstring strain, 391–392
Flexor hinge hand orthoses, 707–709
  external power-driven, 708–709
  passive prehension, 709
  shoulder harness-driven, 708
  wrist extensor-driven, 707–708
Flexor pollicis longus tendon rupture/entrapment, 508–509
Flexor spasms
  in spinal cord injured patients, 190
Flex Sprint, 117–118
Flex Syme's foot, 91, 117–118
Flex Vertical Shock Pylon, 117, 119
Flex Walk, 117–118
Fluid intake
  during immobilization, 751
Fluid resuscitation
  average requirements, 585
  for burn shock, 585–587
  typical programs, 586
Flunarizine, 222
Fluoride, 748
Fluoroscopy
  swallowing study, 327–328
Fluoxetine, 215–217, 220, 476
Fluvoxamine, 216
Flynn phenomenon, 299
FNQ
  See Family Needs Questionnaire (FNQ)
Foam finger pressure wraps, 615
Foam rubber
  in finger prostheses, 39
  for residual limb padding, 61
Follicular hyperkeratosis
  of residual limb, 137
Food and Drug Administration (FDA)
  drug approval, 214
Foot
  arch height, 817
  musculoskeletal disorders, 395–401
  nerve injuries, 531–545
  positioning for burn injuries, 611
  scar compression, 676
Foot amputations
  See Partial foot amputations
Foot blisters, 818
Foot fractures
  stress, 397
Foot orthoses
  for burn injuries, 631, 641–643
  for superficial peroneal neuropathy, 542
Foot prostheses, 89–92
  in above-knee prostheses, 127
  in below-knee prostheses, 97–100, 110, 113
  dynamic response, 92, 115–120, 127
  multiaxis, 114–115, 120
  multiflex, 114–115
  selection, 120
  single-axis, 113–114
  See also specific type of prosthesis
Footwear
  as risk factor for injury, 817
Forced vital capacities (FVCs), 181
Forearm
  scar compression, 673
  strengthening exercises, 376
Forearm orthosis
  with friction joints, 712
Forefoot amputation, 82
Forequarter amputations, 43–45
  prostheses, 59–60, 67
Fort Sam Houston, Texas, 25
Foster Kennedy syndrome, 287
Foster turning frame, 186
Fothergill's disease
  See Tic douloureux
Four bar hydraulic knee joint, 128
Fovea centralis, 286
Foville's syndrome, 296, 311
Fracture cast bracing, 735
Fracture-dislocations

  neuropathies associated with, 440–443
  spinal, 175
Fractures
  brachial plexopathies caused by, 497, 499
  carpal tunnel syndrome caused by, 510, 513
  cranial neuropathies caused by, 280
  early ambulation after, 733–737
  interdisciplinary team management case study, 833–835
  lumbosacral plexopathy caused by, 523–524
  neuropathies associated with, 440–443
  posterior interosseous nerve entrapment caused by, 504
  pronator teres syndrome caused by, 506
  radial neuropathy caused by, 503
  sciatic neuropathy caused by, 525
  spinal, 173–175
  in spinal cord injured patients, 189
  stress, 396–397, 816–818
  ulnar neuropathy caused by, 516
  See also specific type of fracture
Frankel grading
  of incomplete spinal cord injury, 169–170
Frastec, 594
Free flaps, 591–592, 691–692
Free radicals
  and traumatic brain injury, 212
Freezing injury
  caused by volatile liquids, 577
  See also Cold exposure; Frostbite
Fresnel lenses, 299
Frey's syndrome, 303
Friction feeder, 712
Friction joints
  for above-elbow prostheses, 57
Friedreich's ataxia, 217
Frostbite, 123, 595–596, 637, 819–820
  See also Cold exposure
Functional activities
  assistive devices, 682
  for burn patients, 596, 601, 607, 618–621, 631, 674–680
  See also Activities of daily living (ADL)
Functional adaptation complex, 356
Functional assessment
  in traumatic brain injury, 224
  in vocational rehabilitation, 852–853, 855
Functional Assessment Inventory, 852–853
Functional Assessment Measure, 224
Functional biomechanical deficit, 356
Functional capacity
  effect of immobilization on, 743
Functional electrical stimulation (FES), 606–607, 683, 709
  for bracing, 732–733
  energy expenditure for walking with, 733
  for immobility complications, 743–745
  of peroneal nerve, 725–726
Functional Inventory Measure, 224
Functional orthoses
  shoulder/elbow, 713
Functional outcome
  after spinal cord injury, 170–173, 175–176
  after traumatic brain injury, 211–212
Functional substitution
  after traumatic brain injury, 212
Fundoscopy, 288
Furacin, 582
Furosemide, 750
FVCs
  See Forced vital capacities (FVCs)

G

GABAergic agents
  for traumatic brain injury, 213, 216, 219–220, 222
Gabapentin, 477, 490
Gag reflex, 327, 332
Gait pattern
  with knee-ankle-foot orthoses, 729–731
  with metal ankle-foot orthoses, 718–720
  with plastic ankle-foot orthoses, 722–723
  with reciprocating gait orthoses, 732
  swing-through, 178
Gait training
  for lower extremity amputees, 84, 88–89, 98, 102, 104, 106, 126, 133
  for multiple amputees, 149
Gait velocity
  at different levels of lower extremity amputation, 92–93
  with functional neuromuscular stimulation, 733
  in hemiplegic ambulation, 724
  in normal ambulation, 724
  in paraplegic ambulation, 731
  in wheelchair locomotion, 731
Gamma-vinyl GABA
  for traumatic brain injury, 217
Ganglionic blockade
  for causalgia, 489
Gangliosides
  for cranial neuropathies, 291
  for spinal cord injury, 167
Gangrene
  after cold-exposure injury, 123
Gastric fluids
  hypersecretion, 333
Gastric ulcers
  in immobilized patients, 753–755
Gastritis
  stress, 184–185
Gastrocnemius-soleus muscles
  stretching, 398–399
Gastrointestinal complications
  of immobilization, 752–755
  of spinal cord injury, 184–185
Gaze stabilization, 324
GCS
  See Glasgow Coma Scale (GCS)
Gender
  and lifting strength, 806
  and physical capacity for exercise, 803–804
  as risk factor for injury, 816
  See also Women; Men
General Functioning Scale, 254
Geniculocalcarine tract, 286
Genitourinary complications
  of spinal cord injury, 162, 181, 193–194
  See also specific complication
Genucentric knee orthoses, 736
Genu recurvatum, 736
Glasgow Assessment Scale, 224
Glasgow Coma Scale (GCS), 211
Glasgow-Liege Scale, 211
Glasgow Outcome Scale (GOS), 224, 237
Glenohumeral joint injuries, 370–371
Glossopharyngeal breathing (GPB), 182
Glossopharyngeal nerve, 325–329
  anatomy and function, 325–326
Glossopharyngeal neuralgia, 326, 328–329

Glossopharyngeal neuropathies, 326–329
  electrodiagnosis, 328
  evaluation, 327–328
  imaging, 328
  management, 328–329
  prognosis, 328
  symptoms and signs, 327
  syndromes, 326–327
Gloves
  external vascular support, 659, 667
  Isotoner, 616, 618, 658–659, 667
Glucocorticoids
  for musculoskeletal injuries, 357–358, 364
Glucose intolerance
  immobilization-induced, 750–751
Glutamate, 212
Glutamine supplementation, 687
Glycerin suppositories, 753
Glycolysis
  anaerobic, 787–788
Goal setting
  and vocational rehabilitation, 847
Goldthwait, Joel, 830
Gordon Occupational Checklist, 854
GOS
  See Glasgow Outcome Scale (GOS)
Gottfried Medical, Inc., 665
GPB
  See Glossopharyngeal breathing (GPB)
Gradenigo's syndrome, 296, 303
Gradient pressure, 471, 645, 667
  See also Vascular support garments; *specific type of garment*
Graefe disease, 296
Granger, Frank B., 4
Graph-lite prosthetic foot, 115
Grasp
  orthotic substitution for, 704–705
Gray, Barbara, 23
Gray matter, 165–166
  damage, 165
Greissinger foot prosthesis, 114, 120
Grenada, 23
Grieving process
  in lower extremity amputees, 84
Grooming
  adaptive devices, 620, 677–679
Ground electrodes, 453
Growth cone, 432, 434
Guanethidine infusion, 490
Gunshot wounds
  burns secondary to, 581
  cranial neuropathies caused by, 280, 287, 295–296, 326, 339
  spinal cord injuries caused by, 164–165
  traumatic brain injury caused by, 208–209
  See also Penetrating nerve injuries
Gustatory evoked potentials, 314
Gustatory hyperhidrosis syndrome, 312
Gustatory sensation, 310–311
Gustatory sweating, 333
Gustometry, 312
Guttmann, L., 9
Guyon's canal, 514, 519–520
  compression injury at, 520–521
  lesions within, 522

H

Hairs
  coiled, 137
Haldol, 220
Hallucinations
  auditory, 319
  in burn patients, 631
  olfactory, 283
Hallux valgus deformity, 85
Halo skeletal traction, 630
Halstead-Reitan Neuropsychological Battery, 195, 237
Hamilton Rating Scale for Depression, 223
Hamstring injuries, 390–392
Hand
  external vascular support, 658–659
  positioning for burn injuries, 610
  scar compression, 672
Hand amputations, 36–43
  central, 40, 43
  classification, 40
  occupational therapy, 41
  rehabilitation, 37–38, 41–42
Hand centers
  at military hospitals, 9
  World War II, 35, 38
Hand continuous passive motion machine, 616–618, 640–641
Hand contractures
  reduction, 709
Hand controls
  for driving, 54, 64, 178–179
Hand deformities
  caused by burns, 614, 639
Hand function
  with myoelectric prostheses, 69
Hand injuries, 376–378
  World War II, 35
Hand orthoses, 38, 704–709
  adaptive aid attachments, 707
  attachment principles, 704–705
  basic opponens, 705–706
  for burn injuries, 611, 630–631, 639–641
  flexor hinge, 707–709
  grasp assist, 704–705
  long opponens, 706–707
  power sources, 705, 708–709
  with radial extension, 704–705
  reach assist, 704–706, 710–713
  for specific functions, 705–707
  for ulnar neuropathy, 522
Hand prehension patterns, 705
Hand prostheses, 36, 38–43
  cosmetic, 43
Hand reconstruction, 36–37
  after burn injuries, 692–693
  World War II, 35
Hand splint
  short opponens, 705
Hand terminal devices, 53, 70
Hangman's fracture, 174
Harness designs
  for upper extremity prostheses, 55–56
Harris hemisling, 713
Hassles Scale, 253
HDL
  See High density lipoprotein (HDL)
Head injuries
  and alcohol use, 239–241
  associated with spinal cord injuries, 195
  versus brain injuries, 208, 231

    cranial neuropathies caused by, 280, 286, 295, 303, 311, 317, 326, 331, 336, 339
    incidence, 210
    olfactory dysfunction caused by, 282–283
    vertigo after, 319
    vocational rehabilitation, 856
    See also Traumatic brain injury (TBI)
Head orthoses
    for burn patients, 638
    See also Facial orthoses
Head scars
    management, 645–655
Head tilt test, 299
Healing
    delayed, 135
Hearing aids, 324
Hearing impairments, 312, 318–319
    adaptive devices, 324
    causes, 318–319
    incidence, 318
    management, 323–325
    prevention, 325
Hearing loss
    conductive, 318, 323–324
    sensorineural, 318, 324
Hearing scotomas, 319–320
Hearing threshold level (HTL), 318
Heart rate (HR), 790
    exercise training based on, 790
    in immobilized patients, 764
    maximum, 790
    neural control, 790
    response to maximal exercise, 791
Heat cramp, 819
Heat exhaustion, 819
Heating modalities
    used with stretching, 467, 746–747
Heat injuries
    See Thermal injuries; Burn injuries
Heat rash, 819
Heat stress, 818–819
Heat stroke, 819
Heel cutoff
    reduction of knee bending moment by, 717
Heel pain, 536–537
Heelstrike phase
    with metal ankle-foot orthoses, 717–720
    with plastic ankle-foot orthoses, 722–723
Hematoma formation
    after amputation, 134
Hemiinattention
    after traumatic brain injury, 220
Hemipelvectomy, 124
    functional outcome, 83
Hemiplegic ambulation
    energy requirements, 723–724
Hemispatia neglect
    after traumatic brain injury, 220
Hemodynamic changes
    after amputation, 137–139
    in immobilized patients, 764–765
Hemorrhages
    punctate, 209
Hemostasis
    in debrided wound, 588
Henneman's size principle, 457
Heparin, 183, 763–764
Hering-Brewer reflex, 331
Herpes zoster, 296
Hertzman, Carl, 8
Heterograft
    porcine skin, 593
Heterotopic ossification (HO), 188
    neurogenic, 221
Hibiclens
    See Chlorhexidine
High-altitude pulmonary edema, 820
High density lipoprotein (HDL), 182
Highet's grading system, 474–475
High-threshold mechanoreceptors (HTM), 484
Hill-Burton Act Amendments, 6
Hindfoot amputations
    surgical technique, 86–87
    See also Tarsotarsal amputations
Hines Veteran Administration Hospital, Chicago, 162
Hinges
    for above-elbow prostheses, 56–57
    for below-elbow prostheses, 53, 55
    step-up, 44, 53
Hip
    positioning for burn injuries, 610, 631
    scar compression, 675
Hip contractures
    prevention, 101–102, 135
    in spinal cord injured patients, 189
Hip disarticulation, 124
    functional outcome, 83
    socket design, 132–133
    surgical technique, 124
Hip disorders, 389–392
    See also specific disorder
Hip stabilization orthoses, 704
Hip subluxation
    in spinal cord injured patients, 190
Hirschberg reflex, 299
Hispanics
    counseling concerns, 849
Histamine$_2$-receptor antagonists, 754
HIV
    See Human immunodeficiency virus (HIV)
HO
    See Heterotopic ossification (HO)
Hoffman-Tinel sign, 430
Hollister Odor Absorbent dressing, 668
Holmes-Adie syndrome, 287, 297
Homan's sign, 763
Home accessibility aids, 681
Home-based services
    for brain injured patients, 228
Home management
    of burn patient, 680–681
Homograft skin, 593
Hook terminal devices, 53, 70
Horizontal segment
    of facial nerve, 309
Horner's syndrome, 303, 340
Hospital
    vocational assessment in, 850–857
    vocational rehabilitation in, 846–857
Hospital discharge
    vocational rehabilitation after, 858–859
Housekeeping
    adaptive aids, 681
Housing
    for brain injured patients, 227, 229
    for spinal cord injured patients, 180, 196

HR
    See Heart rate (HR)
HTL
    See Hearing threshold level (HTL)
HTM
    See High-threshold mechanoreceptors (HTM)
Hubbard tank, 597
Hughlings-Jackson syndrome, 332
Human immunodeficiency virus (HIV)
    disability cases, 881
Humeral neck amputations, 44–45
    prostheses, 59–60
Humeral rotation devices, 56–57
Hydergine, 219
Hydration status assessment, 751
Hydrocephalus
    communicating, 211
Hydrocolloid dressings, 594
Hydrofluoric acid, 578
Hydrogen-ion homeostasis, 788
Hydronephrosis
    in spinal cord injured patients, 193–194
Hydrostatic weighing, 797–799
5-Hydroxytryptophan, 217
Hygiene
    residual limb, 84, 107–108, 134, 136
Hypafix, 668
Hyperacusis, 312
Hyperalgesia, 482
Hypercalcemia
    immobilization-induced, 750
    in spinal cord injured patients, 185
    treatment, 750
Hypercalcuria, 751
Hyperemia
    nerve damage caused by, 443–444
    reactive, 137
Hyperesthesia, 472
Hyperesthetic desensitization, 472–473, 491–492, 495
Hypermetabolic-catabolic response, 684
Hypermobile segments
    lumbar, 380, 383
Hyperosmia, 283
Hyperpathia, 482
Hyperpigmentation
    of burn wounds, 605, 633
    of residual limb, 136
Hyperplasia
    verrucose, 138
Hyperpyrexia
    after traumatic brain injury, 217
Hyperreflexia
    autonomic, 183–184
Hypersensitivity
    denervation, 435–436, 456
Hypertension
    in spinal cord injured patients, 183–184
Hypertonicity
    definition, 208
    in spinal cord injured patients, 190–191
    See also Spasticity
Hypertrophic scars, 643–645, 655–674
Hyperventilation
    reflex, 789
Hypnosis
    for burn pain management, 622–623
    for phantom limb pain, 147
Hypnotics
    for phantom limb pain, 146
Hypogeusia, 311
Hypoglossal nerve, 338–342
    anatomy and function, 338–339
    stimulation, 340–341
Hypoglossal neuropathies, 339–340
    electrodiagnosis, 340–341
    imaging, 341
    management, 341–342
    prognosis, 341
    symptoms and signs, 340
    syndromes, 340
Hyposmia, 283
Hypotension
    orthostatic, 175, 177, 184
Hypothermia, 819
    and traumatic brain injury, 213
Hypoxic brain injury, 211, 234–235

I

IASP
    See International Association for the Study of Pain (IASP)
IC
    See Intermittent catheterization (IC)
Ice
    See Cryotherapy
Icelandic-New York (ISNY) socket, 130
Ileus
    in spinal cord injured patients, 184
Iliotibial band (ITB) syndrome, 389–390
Ilizarov technique
    of stump lengthening, 96
Imaging studies
    in accessory neuropathies, 338
    in glossopharyngeal neuropathies, 328
    in hypoglossal neuropathies, 341
    in musculoskeletal injuries, 381–382
    in oculomotor neuropathies, 299–300
    in olfactory neuropathies, 285
    in spinal cord injury, 163–164, 173, 175, 192
    in traumatic brain injury, 211, 225, 230–231
    See also specific type of study
Imipramine, 476
Immediacy
    principle of, 623
Immediate postoperative prosthesis (IPOP)
    advantages and disadvantages, 98
    construction, 96–98
    for lower extremity amputations, 88, 96–98, 100, 124, 126
    for upper extremity amputations, 48
    and wound breakdown, 99–100
Immediate postoperative rigid dressing (IPORD)
    advantages and disadvantages, 99
    for lower extremity amputations, 87–88, 96, 99, 103–106, 124–126
    weight bearing, 99–100, 102
    and wound breakdown, 99–100, 135
Immersion foot, 443–446
Immobility complications, 741–777
    cardiovascular, 762–765
    endocrine, 750–751
    functional electrical stimulation for, 743–745
    gastrointestinal, 752–755
    historical background, 742
    integumentary, 755–758
    management, 743–765
    metabolic, 750–751

  monetary impact, 742
  musculoskeletal, 356, 359, 743–765
  neurologic, 748–750
  pathophysiology, 743
  Persian Gulf War, 742, 745, 756
  prevention, 8–9, 84, 101, 226
  respiratory, 758–762
  urinary tract, 751–753
  Vietnam War, 742
  See also Pressure sores; Contractures
Immobilization
  for back injuries, 383
  complications caused by, 102, 135, 175
  contractures during, 745–746
  for musculoskeletal injuries, 358–359, 371–372, 378, 385
  postgraft, 627, 629
  therapeutic use, 743
  types, 743
Immobilization orthoses
  postgraft, 629–631
Immobilization osteoporosis, 747–748, 750
Immobilization phase
  of wound care, 595–596, 625–631
Impingement syndrome
  in spinal cord injured patients, 189
Inactivity
  See Immobilization
Incentive spirometry, 759–760
Incontinence
  in spinal cord injured patients, 193
Independent living
  for brain injured patients, 227, 229
  for spinal cord injured patients, 180, 196
Indomethacin, 354
Induction Medical Form, 869–871
Infantry
  musculoskeletal injuries rate, 815–816
Infection
  after amputation, 134
Inferior vena cava filters, 183
Inflamed joints
  immobilization of, 743
Inflammatory reaction, 354, 357
Inhalation analgesics
  for burn patients, 622
Inhalation injury, 584–585, 598–599, 620
Innervation
  muscular, 784–785
  vascular, 428
Innervation ratio, 448–449
Inserts
  for orthoses, 650, 664–674
Inspiration
  factors affecting, 181–182
Insulin, 185
Integumentary system
  See Skin
Intensity
  of training, 807, 810, 812–813
Interdigital neuromas, 544–545
Interdigital web spacers, 615–616, 640, 642
Interdisciplinary team, 7, 829–844
  Army, 16
  case studies, 833–844
  in combat theater, 8–9
  composition of, 831–833
  concept of, 6–7
  historical perspective, 830–831
  for lower extremity amputation, 81, 85, 89, 92, 101, 127, 133, 147–148
  for spinal cord injury, 175, 180, 195
  for traumatic brain injury, 208, 225–229, 255
  for upper extremity amputation, 47
  World War II, 9–10
Interdisciplinary team conference, 830
Interest assessment
  in vocational rehabilitation, 854–855
Interest Check List, 854
Interference pattern, 458
Intermittent catheterization (IC)
  in spinal cord injured patients, 193
Intermittent mandatory ventilation mode, 599
International Association for the Study of Pain (IASP), 481
Interphalangeal (IP) joint
  amputation at, 37
  extension attachments, 706
Interphalangeal (IP) joint stabilizers, 709
Inter-potential interval (IPI), 463
Interview
  for neurobehavioral assessment, 242–243
  for vocational assessment, 853
Intracompartmental pressure, 440
Intrafascicular derangement, 430
Intrinsic bar
  long opponens splint with, 706–707
Intubation
  of burn patients, 584, 598–600
Inversion sprains
  ankle, 397–399
Investigation report
  line of duty (LOD), 868–869, 877
IOMED, 66
Ion channels
  cell membrane, 423–424
Iowa City Buckner Microstomia Prevention Appliance (MPA), 615–616
IPI
  See Inter-potential interval (IPI)
IPOP
  See Immediate postoperative prosthesis (IPOP)
IPORD
  See Immediate postoperative rigid dressing (IPORD)
Iraq
  See Persian Gulf War
Ischemia
  residual limb pain caused by, 139
Ischemic brain injury, 211, 234–235
Ischemic cord injury, 166
Ischemic neuropathy, 439–440
Ischial containment sockets, 129–130
Ischial weight-bearing orthoses, 734–735
Ishihara's chart, 288
ISNY socket
  See Icelandic-New York (ISNY) socket
Isokinetic dynamometer, 802–803
Isokinetic exercises, 813
  for musculoskeletal injuries, 360
  for peripheral neuropathy, 470
Isokinetic fatigue test, 801
Isometric contractions, 783
Isometric exercises, 813
  for immobility complications, 743
  for musculoskeletal injuries, 360, 362, 369, 371–374, 377
  for peripheral neuropathy, 469
Isotoner gloves, 616, 618, 658–659, 666–667
Isotonic compression gloves, 471

Isotonic exercises, 813
  for musculoskeletal injuries, 360, 362, 393
  for peripheral neuropathy, 469
Israeli Defense Force
  combat injuries, 13–16, 72, 150, 162
  exertion-related deaths, 817
  stress fractures, 817
Isshiki thyroplasty, 335
ITB syndrome
  See Iliotibial band (ITB) syndrome
Itching
  after burn injury, 633

J

Jackson’s syndrome, 340
Jacob’s ladder effect, 578
Jaeger cards, 288
Jay cushion, 196
Jay Medical Ltd., 196
Jefferson fracture
  C-1, 173–174
Jeltrate, 654
Jitter, 463
Job coach
  for brain injured patients, 248–250
Job search assistance, 858
Job site training/enabling
  for brain injured patients, 249
Jobst custom stockings, 660
Jobst Institute, Inc., 665
Jobst Interim glove, 659
Jobst Intermittent Compression Pump, 606–607, 682
Jogger’s foot, 534, 537
Joint capsule tightness
  in spinal cord injured patients, 189
Joint contractures
  prevention, 88, 101–102, 125–126, 135
  in spinal cord injured patients, 189
Joint immobilizers
  prefabricated, 358
Joint mobility, 387
  passive resistance, 172
  See also Range-of-motion exercises
Joints
  burn injury-induced changes, 690
  inflamed, 743
  preservation, 83
  See also specific joint
Jones, Sir Robert, 20
Joule’s law, 579
Joystick control
  of wheelchair, 177
Jugular foramen syndrome, 326, 332

K

KAFOs
  See Knee-ankle-foot orthoses (KAFOs)
Kainate, 212
Kanamycin, 591
Kaneda instrumentation, 175
Keloid, 644
Kerlix, 600, 613, 630, 645
Ketorolac, 495
Kevlar shank-ankle-foot unit, 116
Kevlar socks
  for foot prostheses, 116
Keyboard usage
  adaptive aids, 681–682
Kinaire bed, 604, 608, 628
Kinaire foot cushion, 611
Kinesiotherapist, 831
Kinesthetic sensations
  phantom limb, 143
Kinetic Concepts, 182, 186
Kinetic sensations
  phantom limb, 143
Kinnairbed, 186
Kirk, Norman T., 38
Kirkaldy-Willis’ degenerative cascade, 379
Kitchen activities
  adaptive aids, 681
Klenzak-type ankle joint, 716–717
Kling, 600, 630
Kluver-Bucy syndrome, 222
Knee
  bending moment at, 717, 723
  fixed/locked, 734–735
  joint salvage, 93, 123
  positioning for burn injuries, 610–611
  scar compression, 675
  valgus deformity, 736–737
  varus deformity, 736–737
Knee alignment
  orthoses for, 736–737
Knee-ankle-foot orthoses (KAFOs), 178, 726–733
  anterior stops, 729
  bilateral, 178, 731–732
  components, 726–729
  knee stabilization force, 726–727
  metabolic requirements, 729
  pelvic bands, 730–731
  reciprocating gait, 732
  Scott-Craig, 729, 732
  shear forces, 727–729
  standing balance, 730
  types, 726–727
Knee cage designs, 736
Knee continuous passive motion machine, 618
Knee contractures
  prevention, 135
  in spinal cord injured patients, 189
Knee disarticulation, 82, 122–124
  advantages, 122
  functional outcome, 83
  indications, 124
  prostheses, 83, 123
  surgical technique, 124
Knee dislocations
  stretch neuropathies associated with, 440–441
Knee disorders, 392–395
  ligament, 355, 392–395
  See also specific disorder
Knee lock
  manual, 128
Knee orthoses
  genucentric, 736
Knee prostheses, 127–133
  biomechanically stable, 128
  brake mechanisms, 127–128
  classification, 128
  disarticulation, 83
  fabrication, 129–131
  polycentric (four-bar), 128
  single axis, 128
  sockets, 129–131

  stability of, 127–128
  suspension, 131
Knee shear
  with knee-ankle-foot orthoses, 727–729
Knee stability
  with ankle-foot orthoses, 720, 723
  with knee-ankle-foot orthoses, 726–727
Knuckle-bender orthosis, 709
Korean War
  amputations, 134
  physical therapy, 22–23
Krukenberg procedure, 64
Krusen, Frank H., 5, 8
Kuehlthau, Brunetta A., 22
Kushion flex, 670
Kuwait
  See Persian Gulf War

L

Labyrinthine segment
  of facial nerve, 309
Lachman test, 392
Lacrilube, 315
Lacrimation
  dysfunction, 312
Lactate threshold, 789
Lactic acid accumulation, 787, 789
Lamb's wool, 668
Lamotrigine, 222
Language disorders
  after traumatic brain injury, 222
Larrey, Barron, 443
Laryngeal nerves
  evaluation, 333–334
  muscles supplied by, 331
  paralysis, 331–332
  stimulation, 330
Laryngoplasty, 335
Laryngoscopy, 333
Laser beam scanning
  for socket design, 121
Latency, 459
  residual, 512
Lateral geniculate body, 286, 289
Lateral plantar nerve, 532–533
  branch-I entrapment, 536–537
Lateral rotational treatment
  for pulmonary complications, 759
Lawson General Hospital, Georgia, 35, 80
L-dopa
  See Levodopa
Leak speech, 181
LE amputations
  See Lower extremity (LE) amputations
Leave
  convalescent, 872
Lebanese Civil War, 14–15, 482, 490
Lecithin, 218
Leeds anxiety and depression scales, 251, 253
Legal issues
  with traumatic brain injury, 235
Legal representation
  during physical disability review process, 875
Leg amputations
  See Lower extremity (LE) amputations
Leg disorders
  musculoskeletal, 395–401
  See also specific disorder
Lehmann, Justus F., 6
Levator palpebrae muscle
  innervation, 292–293
Levodopa, 214–215, 219, 222
Lichenification
  of residual limb skin, 136–137
Lidocaine, 361, 372, 382, 513
Lifting strength
  gender differences, 806
  measurement, 806
Ligament injuries, 355, 392–394
  spinal, 173–174
  See also specific injury
Ligament of Struthers, 506
Ligaments
  function, 355
  resistance training-induced changes, 810
Limb length preservation, 83, 95–96
Limb salvage
  versus lower extremity amputations, 81–83, 122–123
Line of duty (LOD)
  disability payments, 865
  investigation report, 868–869, 877
Liners
  for prosthetic sockets, 107–108, 140–141
Lingual nerve
  compromise, 304
Lipid, 792
Lipid intake
  and burn injuries, 687
Lisfranc's amputations
  See Tarsometatarsal amputations
Lisuride, 214–215
Lithiasis, 751
Lithium carbonate, 240
LLB
  See Long leg braces (LLB)
LMB finger pressure wraps, 612–613, 615, 658
Load cells, 802
Local anesthetic blocks
  for phantom limb pain, 146
Local flaps, 691
Locked ankle orthoses, 734–735
Lockjaw, 304
LOD
  See Line of duty (LOD)
Longitudinal arch, 537
  medial plantar nerve entrapment at, 537–538
Long leg braces (LLB), 733
Long's Line, 129
Long-term care facilities
  for brain injured patients, 226–227
Long thoracic nerve, 496
  injury to, 498
Lorazepam, 220
Low-air-loss bed, 186, 757
Low back pain, 11, 378–389, 857
Lower extremity (LE) amputations, 79–159
  causes, 34
  complications, 134–148
  early, 122–123
  and energy expenditure for walking, 92–93
  indications, 122
  level selection, 83, 135
  versus limb salvage, 81–83, 122–123
  multiple, 148–149
  nerve recovery in, 82

  patient education, 84, 101–102
  rehabilitation, 81–85, 93, 101, 106
  sensory feedback, 85, 93
  surgical procedures, 83–84
  World War II, 34–35
  See also Above-knee amputations (AKA); Below-knee amputations (BKA); *specific type of amputation*
Lower extremity nerve injuries, 522–531
  See also specific injury
Lower extremity orthoses, 704, 715–737
  See also specific type of orthosis
Lower extremity prostheses
  long-term maintenance, 92, 106, 133–134
  postoperative fitting, 85
  psychological support, 84
  residual limb interface, 89, 96–97, 104, 107, 129, 131, 136–137, 142–143
  temporary, 84, 88, 96–100
  See also specific type of prosthesis
Lower extremity scar compression, 675–676
Lower respiratory tract infection
  in immobilized patients, 759
Low load
  definition, 603
Low threshold mechanoreceptors (LTM), 484
Lumbar plexus, 522–523
Lumbar radiculopathy, 379–381
Lumbar spine
  contusions, 386–389
  Kirkaldy-Willis' degenerative cascade, 379
  muscle strain, 386–389
  pars interarticularis injury, 385–386
  posterior element pain, 384
Lumbar spine disorders, 378–389
  classification, 378–382
  diagnosis, 378–382
  imaging, 381–382
  rehabilitation, 382–385
Lumbar sympathetic blockage
  for causalgia, 489–490
Lumbosacral plexopathy, 522–524
  anatomic considerations, 522–523
  clinical presentation, 523
  electrodiagnosis, 523–524
  etiology, 523
Lumbosacral spine, 167–168
  injury, 169
Lumbrical bar hand orthosis
  for ulnar neuropathy, 522
Lund and Browder chart, 580
Luria-Nebraska Neuropsychological Battery, 195

M

Maceration
  of residual limb skin, 136
Macfarlane microstomia correction device, 638
Macrophages
  role after denervation, 433
Madigan Army Medical Center, Washington, 10–11
Mafenide acetate, 582
Magnesium depletion
  immobilization-induced, 750
Magnetic resonance imaging (MRI)
  in cranial neuropathies, 285, 299, 341
  in musculoskeletal injuries, 381
  neuroma location with, 140
  socket design with, 121, 130
  in spinal cord injury, 163–164, 173, 192
  in traumatic brain injury, 225, 230
Makeup
  for burn patients, 673, 690
Mallet finger, 614
Mandibular nerve, 300
Mangled Extremity Syndrome Index (MESI), 82, 122–123
Mannitol, 585
Marcus-Gunn pupil, 290
Mare Island Naval Hospital, California, 10, 52, 149
Marine Corps
  body fat standards, 796–798
Marital instability
  and brain injuries, 253–254
  and spinal cord injured patients, 196
Martin-Gruber anastomosis, 517–518
Massage
  desensitization with, 634
  external, 471
  for phantom limb pain, 147–148
Massage and Therapeutic Exercise (McMillan), 21
Masseter inhibitory reflex
  electrodiagnostic evaluation, 305
Masseter reflex, 302
  electrodiagnostic evaluation, 305
Master knot of Henry
  See Longitudinal arch
Mastication muscles
  paralysis, 304–305
Mastoid segment
  of facial nerve, 309
Maturation phase, 355
Maxillary nerve, 300
Maximum physiologic potential, 743
Maximum voluntary isometric contraction (MVC), 744
Mayo Clinic, 5, 9
McCance, R. A., 742
McCloskey General Hospital, 35, 80
McGuire General Hospital, Virginia, 80
MCL
  See Medial collateral ligament (MCL)
McMaster Family Assessment Devise (FAD), 252, 254
McMillan, Mary, 20–22, 830
MCP joint
  See Metacarpophalangeal (MCP) joint
MCVOST
  See Medical College of Virginia Olfactory Screening Test (MCVOST)
Meatal segment
  of facial nerve, 309
MEB
  See Medical Evaluation Board (MEB)
Mechanical ventilation
  in burn patients, 599
MEDCOM
  See Army Medical Command (MEDCOM)
Medial antebrachial cutaneous nerve injury, 498–499
Medial capsuloligamentous injuries, 374–376
Medial collateral ligament (MCL), 393–394
Medial cutaneous nerve, 496
Medial plantar nerve, 532–533, 543
  entrapment at longitudinal arch, 537–538
Medial tibial stress syndrome, 395–396
Median nerve, 496, 506–507, 509–510
Median neuropathy, 505–514
  case scenario, 491–493
  compression sites, 505–506
  orthotics, 640–641

Medical College of Virginia Olfactory Screening Test (MCVOST), 284
Medical diagnoses
  and disability compensation evaluation, 872
Medical Evaluation Board (MEB), 865–868
  establishment, 868
  membership, 868
  Narrative Summary (NARSUM), 868–872
  versus Physical Evaluation Board (PEB), 872
Medical Force 2000 (MF2K), 28–29
Medical history of patient
  in disability compensation evaluation, 868–869
Medical treatment facilities (MTFs)
  brain injury rehabilitation centers, 225–226
  physical medicine and rehabilitation at, 9–16
  spinal cord injury treatment at, 163
  See also specific center
Medicare/Medicaid, 6
Medications
  See Pharmacologic therapy; *specific medication*
Medroxyprogesterone acetate, 222
Medulla, 790–791
Megacolon
  in spinal cord injured patients, 185
Melanocyte stimulating hormone (MSH), 218
Mellaril, 220
Membrane instability
  denervation, 435–436
Membrane potential, 423–425, 449
  alteration following denervation, 436
Memory function
  and neurotransmitter systems, 218
  and traumatic brain injury, 218, 230
Men
  military body fat standards, 796, 798
  percent body fat values, 794
  physical capacity for exercise, 803–804
  reference body composition values, 792
Menace reflex, 313
Mendez, Cristobal, 742
Mental concentration
  during exercise periods, 635
Mental status alterations, 230
Meperidine, 495
Mephanesin, 308
Merthiolate, 632, 656
Mesencephalon, 291–292
Meshed skin grafts, 589–591, 644
MESI
  See Mangled Extremity Syndrome Index (MESI)
Metabolic requirements
  of burn patients, 684–686
  See also Energy expenditure for walking
Metabolism
  effect of immobility on, 742, 750–752
Metacarpals
  phalangization, 36
Metacarpophalangeal (MCP) joint
  amputation at, 37, 42
  extension attachments, 705–706
Metal orthoses
  ankle-foot, 715–720
Metamucil
  See Psyllium hydrophilic mucilloid fiber
Methadone, 495, 600, 622
N-methyl *D*-aspartate, 212
Methyldopa, 220
Methylphenidate hydrochloride, 214–215, 219
Methylprednisolone, 163, 167, 513
Metropolitan Life Insurance tables, 793, 797
Mexiletine, 146, 148, 477–478
MF2K
  See Medical Force 2000 (MF2K)
Microfoam tape, 670
Microstomia correction devices, 638
Microstomia Prevention Appliance (MPA), 615–616
Microwave diathermy, 746
Midazolam, 622
Midfoot amputations, 85
  prosthetic fitting and training, 90
  surgical technique, 86
  See also Tarsometatarsal amputations; Transmetatarsal amputations
Military history of patient
  in disability compensation evaluation, 868–869
Military Medical Retention Board (MMRB), 867
Military Occupational Specialty-Military Medical Retention Board (MOS/MMRB), 867
Military pensions
  See Physical disability system
Military performance
  physical fitness and training for, 781–827
Military populations
  body fat percentage values/standards, 794
  physical capacity for exercise, 803–806
Military services
  body fat measurement methodology, 797–799
  body fat standards, 796–797
  physical disability system, 863–885
  weight control programs, 793, 796–797
Millard-Gubler syndrome, 296, 311
Mind-body connection, 846
Mineral requirements
  of burn patients, 687
Mini compartment syndrome, 540
Minnesota Multiphasic Personality Inventory (MMPI-2), 195, 239, 241, 244–245, 857
Minority groups
  counseling concerns, 849
Miosis
  in darkness, 299
Misdirection syndrome, 299
Misoprostol, 754–755
Missile wounds
  brachial plexopathies caused by, 497
  spinal cord injuries caused by, 164
  traumatic brain injury caused by, 208–209
Mithramycin, 750
MMPI-2
  See Minnesota Multiphasic Personality Inventory (MMPI-2)
MMRB
  See Military Medical Retention Board (MMRB)
MMTN
  See Myelinated mechanothermal nociceptors (MMTN)
Mobility training
  for lower extremity amputees, 84, 102
Mobilization
  after spinal cord injury, 175
  See also Ambulation
Möbius' syndrome, 296, 311
Modular Flex Foot, 117–118
Moleskin, 668
Monopolar needle electrodes, 453
Morphine, 217, 600
Morphine sulfate, 495

Morton’s toe neuroma, 543
MOS/MMRB
    See Military Occupational Specialty-Military Medical Retention Board (MOS/MMRB)
Motion Control, Inc., 66
Motivation
    to recover, 846–847
Motoneurons, 449–450, 784–785
Motoneuron weakness
    after spinal cord injury, 167
Motor cortex, 790
Motor fitness, 782
Motor function
    after peripheral neuropathy, 464–465
    and ulnar neuropathy, 516
Motor unit, 448–449, 784–785
    activity duration, 456
    electrical morphology, 456–457
    recording phases, 456–457
    recruitment, 457–458, 785
    size, 448–449
Motor unit potential
    amplitude, 457
    polyphasic, 456–457, 463–464
Mouth
    positioning for burn injury, 609
Mouth stretch
    for facial contractures, 652
Mouth-stretching devices, 652–653, 655
Movement disorders
    after traumatic brain injury, 220–221
MP
    See Myoelectric prostheses
MPA
    See Microstomia Prevention Appliance (MPA)
MRI
    See Magnetic resonance imaging (MRI)
M.S. Contin, 622
MSH
    See Melanocyte stimulating hormone (MSH)
MTFs
    See Medical treatment facilities (MTFs)
Mucomist
    See Acetylcysteine
Muenster socket, 53
Multiaxis foot prostheses, 114–115, 120
Multidisciplinary approach
    See Interdisciplinary team
Multiflex ankle/foot, 114, 120
Multiple sclerosis, 296, 322
Multireceptive neurons, 484
Muscle
    denervation-induced changes in, 434–436
Muscle(s)
    antagonist, 782
    artificial, 708–709
    contractility, 783–784
    energy stores, 787
    energy transformation within, 786
    fiber types, 784
    force development, 785
    immobility-induced ultrastructural changes in, 744–745
    innervation, 784–785
    oxidative capacity, 801
    and physical fitness, 792–799
    structure and function, 782–786
Muscle atrophy
    after denervation, 435
    in burn patients, 606
    due to immobilization, 744–745
Muscle fatigue, 786
Muscle fibers
    classification, 784
    contractile tension loss in, 786
    fast twitch (type 2), 784, 802, 809
    slow twitch (type 1), 784
    structure, 783
    type distribution, 784
Muscle hypertrophy, 809
Muscle mass
    and body weight, 793
    correlation between body strength and, 795–796, 803
    increase in, 793–794
    loss in, 794
Muscle sites
    for myoelectric prostheses, 69
Muscle soreness
    avoidance, 603
Muscle strain
    lumbar, 386–389
Muscle strength
    See Strength
Muscle tone
    grading, 172, 208
Muscular contraction
    concentric, 783
    eccentric, 783
    isometric, 783
    mechanism, 783–784
    neural control, 784–786
    static, 783
    types, 782–783
Muscular endurance
    See Anaerobic power
Musculocutaneous flaps, 691–692
Musculocutaneous nerve injury, 499
Musculoskeletal disorders, 353–356
    components, 356–357
    diagnosis, 356–357
    exercise-associated, 814–817
    forward care, 8, 11–13, 24
    healing, 355–356, 358–360
    local effect minimization, 357–358
    mechanisms, 354–355
    pathophysiology, 354–356
    Persian Gulf War, 11–13
    physical therapy for, 20, 24
    rehabilitation, 8, 11–13, 29, 356–401
    return to duty, 360
    in spinal cord injured patients, 188–189
    symptoms and signs, 355–356
    World War I, 354
    See also specific disorder
Musculoskeletal system
    effect of immobility on, 743–748
Mustard, 578
MVC
    See Maximum voluntary isometric contraction (MVC)
Myasthenia gravis, 296
Myelin, 422–423
Myelinated mechanothermal nociceptors (MMTN), 484
Myelin debris removal
    role of macrophages in, 433
Myelin sheath
    denervation-induced changes in, 433
Myelin thickness

  - and conduction velocity, 449
- Myelograms, 381
- Myocardial infarction
  - silent, 184
- Myoclonus
  - after traumatic brain injury, 220–221
- Myocutaneous flaps
  - for pressure sores, 187
- Myodesis, 95, 124
  - definition, 122
  - *versus* myoplasty, 122
  - unbalanced, 140
- Myoelectric electrodes, 65, 67
- Myoelectric prostheses
  - above-elbow, 56
  - advantages and disadvantages, 69, 71
  - below-elbow, 53
  - bilateral, 62
  - components, 67–68
  - control systems, 65–67
  - *versus* conventional prostheses, 68–71
  - cosmetic covers for, 68–69, 71
  - evaluation of candidates, 68
  - history, 65
  - humeral neck, 59
  - hybrid (myoelectric and body-powered), 62
  - rehabilitation issues, 68–69
  - sensory feedback with, 67, 69
  - for upper extremity amputations, 44–46, 65–69
  - Utah brand, 67–68
- Myoelectric testing device, 69
- Myofibrils, 783–784
- Myofibroblasts
  - and hypertrophic scar formation, 644
- Myofilaments
  - denervation-induced loss, 434–435
- Myogenic contractures, 746
  - in denervated muscles, 435, 465–467, 491
- Myokymic discharges, 455
- Myoplasty, 95
  - definition, 122
  - *versus* myodesis, 122
- Myosin, 783–784
- Myositis ossificans
  - *See* Heterotopic ossification (HO)
- Myotendinous contracture
  - in spinal cord injured patients, 189
- Myotomal levels
  - for neurological evaluation of spinal cord injury, 169

N

- NAD$^+$
  - *See* Nicotinamide adenine-dinucleotide (NAD$^+$)
- Naftidrofuryl, 219
- Naloxone, 216
- Naltrexone, 216–217
- Naprosyn, 492, 495
- Narcotics
  - for musculoskeletal injuries, 364
  - for phantom limb pain, 146, 148
  - for spinal cord injured patients, 191–192
- Narrative Summary (NARSUM)
  - Medical Evaluation Board (MEB), 868–872
- National Center for Medical Rehabilitation Research, 6
- National Head Injury Foundation (NHIF), 227
- National Institutes of Health, 6
  - obesity definition, 793, 797
- Navy
  - body fat standards, 796–798
  - disability system, 883–884
  - obesity definition, 793, 797
  - physical therapy services, 25–26
- Neck
  - contracture prevention, 630, 638, 649–652
  - passive stretches for, 362
  - positioning for burn injuries, 609
  - scar management, 645–655
  - strengthening, 362
- Neck and tongue syndrome, 303
- Neck injuries, 361–363
  - cranial neuropathies caused by, 280, 336
  - *See also specific injury*
- Neck orthoses, 630, 638, 650–652
- Necrosis
  - after amputation, 135
- Necrotic muscle
  - left in wound, 81
- Needle electrodes, 453
  - concentric, 453
  - monopolar, 453
  - single fiber, 453
- Negative feedback loop
  - in musculoskeletal injuries, 357
- Neoprene, 670
- Nerve blocks
  - for lumbar pain, 382
  - for residual limb pain, 147
- Nerve conduction block
  - electrodiagnostic findings, 460–461
- Nerve conduction studies (NCS), 458–459
  - in accessory neuropathies, 338
  - action potential duration, 459
  - amplitude, 459
  - in anterior interosseous syndrome, 509
  - in brachial plexopathies, 498–500
  - in carpal tunnel syndrome, 512–513
  - in compartmental syndromes, 440
  - conduction velocity, 459
  - and determination of injury severity, 459–462
  - electrodes, 453
  - in facial neuropathies, 314–315
  - in femoral neuropathy, 526–527
  - filter settings, 452
  - in foot and ankle nerve injuries, 531
  - in hypoglossal neuropathies, 340–341
  - instrumentation, 450–453
  - latency, 459
  - limitations, 464–465
  - localization with, 462
  - in lumbosacral plexopathy, 524
  - in peroneal neuropathy, 529–530
  - in posterior interosseous nerve entrapment, 505
  - in pronator teres syndrome, 508
  - in radial neuropathy, 503
  - recovery monitoring, 463–464
  - in sciatic neuropathy, 525–526
  - in superficial radial nerve compression, 505
  - in sural entrapment neuropathy, 543
  - in tarsal tunnel syndrome, 535
  - timing of, 460–461
  - in ulnar neuropathy, 514, 517–522
  - in vagus neuropathies, 333–334
- Nerve conduction velocity calculation, 459
  - effects of temperature on, 445
  - and myelin thickness, 449

Nerve entrapment
  peripheral, 192–193
Nerve excitability studies
  in facial neuropathies, 314
Nerve fibers
  myelinated, 422–423
  unmyelinated, 422–423
Nerve impulse propagation, 424–425, 449–450, 785
Nerve root stimulation
  for thoracic outlet syndrome, 502
Nerve tissue pressure thresholds, 438
Nerve trunk pain, 475
Nerve trunk transection, 431
Neural adaptation
  in resistance training, 810
Neural connection alterations, 212
Neuralgia
  atypical, 280
  trigeminal, 280
Neural membrane stabilizers
  for peripheral nerve injuries, 477–478, 490
Neural recovery mechanisms, 212–213, 431–432
Neural reserve
  and brain injury prognosis, 210
Neuroanatomy, 422–423, 426–427
Neurobehavioral assessment, 231, 237
  protocol, 242–244
  test interpretation and reporting, 244–245
  test selection and administration, 243–244
  theoretical rationale, 241–242
Neurobehavioral programs
  for brain injured patients, 228
Neurobehavioral Rating Scale, 223
Neurogenic heterotopic ossification
  after traumatic brain injury, 221
Neurogenic spinal arthropathy
  See Charcot's spine
Neuroimaging
  in traumatic brain injury, 211, 225, 230–231
Neuroleptics
  for phantom limb pain, 146
  for traumatic brain injury, 217, 220, 240
Neurological complications
  of burn injury, 689–690
  of immobility, 748–750
  of spinal cord injury, 189–193
Neurological evaluation, 167–173, 195
  myotomal levels, 169
  segmental reflexes, 171–172
  sensory levels, 169
Neurologic states
  low level, 234–235
Neuromas
  acoustic, 322
  in lower extremity amputees, 122, 140
  and phantom limb pain, 144, 146–147
  in upper extremity amputees, 61
Neuromatrix theory
  of phantom limb pain, 126, 145
Neuromedical issues
  with traumatic brain injury, 217, 222–224
Neuromedical workup
  standard, 227
Neuromuscular junction, 425, 450, 784–786
  failure, 460
Neuromusculoskeletal conditions
  physical therapy for, 20, 24–26
  See also Musculoskeletal disorders; *specific disorder*
Neuron, 422–423
  motor, 449–450, 784–785
  multireceptive, 484
  vascular supply, 427–428
Neuronal reorganization, 212
Neuronal signaling, 423–425
Neuronal sparing, 212–213
Neuropathic pain
  classification, 475
  management, 475–479
Neuropathy
  diabetic ischemic, 296
  evaluation, 82
  Persian Gulf War, 11–14, 38, 80
  See also Cranial neuropathies; Peripheral nerve injuries; Spinal cord injury (SCI); *specific injury*
Neurophysiology, 212–213, 423–426, 448–450
  of peripheral nerves, 448–450
Neuropraxia, 429
Neuropsychological tests
  interpretation and reporting, 244–245
  selection and administration, 243–244
  See also specific test
Neuropsychopharmacology, 214–222
Neurosignature, 145
Neurotmesis, 429
Neurotoxic cascade
  after traumatic brain injury, 212–213
Neurotransmitters, 425, 450, 785
  and traumatic brain injury, 212–213, 218
NHIF
  See National Head Injury Foundation (NHIF)
Nicardipine, 479
Nicotinamide adenine-dinucleotide (NAD$^+$), 786
Nifedipine, 183–184, 479
97th General Hospital, Germany, 11
Nitrofurantoin, 752
Nitrogen balance equations, 688, 750
Nitropaste, 183
Nizatidine, 754
NMS conditions
  See Neuromusculoskeletal conditions
Nodes of Ranvier, 423
Nonphysician healthcare providers, 24–26
  See also specific type of provider
Nonsteroidal antiinflammatory drugs (NSAIDs)
  adverse effects, 193
  for musculoskeletal injuries, 354, 357, 367, 383
  for peripheral nerve injuries, 478–479, 490
  for phantom limb pain, 145, 148
  for traumatic brain injury, 221
  See also specific drug
Nootropics
  for traumatic brain injury, 219
  See also specific drug
Noradrenergic treatment
  for traumatic brain injury, 213, 220
Norepinephrine, 212, 219, 476
Normal shape, normal alignment (NSNA) socket, 129, 139
Northwestern University, 5, 9
Nortriptyline, 219, 476
Nose
  positioning for burn injury, 609
  prosthetic, 655
Nose orthosis, 655
NSAIDs
  See Nonsteroidal antiinflammatory drugs (NSAIDs)
N-ter-face, 591, 594

Numb cheek syndrome, 303–304
Numb chin syndrome, 304
Nurses
    role in interdisciplinary team, 832–833
    World War II, 22
Nursing
    during acute phase of burn injury, 596–607
    during immobilization phase of burn injury, 625–627
    during wound maturation phase of burn injury, 632–635
Nutrition
    and amputation complications, 135
    and burn patients, 602, 684–689
    and cranial neuropathies, 285, 296
    and immobilization, 752, 754
    and pressure sores, 187
Nutritional assessment
    of burn patients, 684–687
    of immobilized patient, 752
Nutritional monitoring
    of burn patients, 688–689
Nutrition care planning
    for burn patients, 687–688
Nystagmus, 319, 321

O

Oakland Naval Hospital, 149
Ober's test, 390
Obesity, 793
    Navy definition, 793, 797
OBLA
    See Onset of blood lactate accumulation (OBLA)
Obstipation
    in spinal cord injured patients, 185
Occulocardiac reflex, 333
Occupation
    and aerobic fitness, 804
    and disability compensation evaluation, 869–872
    and lower back pain, 378–389
    physical training specific to, 808, 814
Occupational therapists
    Korean War, 23
    role in interdisciplinary team, 7, 831–832
Occupational therapy
    for above-knee amputees, 133
    in Army, 16
    for functional impairments, 15
    history, 830–831
    importance of, 8–9
    for lower extremity amputations, 84, 102
    for mild traumatic brain injury, 231
    for multiple amputees, 149
    for nerve injuries, 12
    for spinal cord injured patients, 179–180
    for upper extremity amputations, 38, 41–42, 52
    See also Vocational rehabilitation
Occupational Therapy Association, 831
Ocular muscles
    innervation, 293–294
Ocular neuromyotonia, 297
Oculomotor nerve, 291–300
    anatomy and function, 291–295
    muscles innervated by, 293–294
Oculomotor neuropathies, 295–300
    electrodiagnosis, 299
    imaging, 299–300
    management, 299–300
    symptoms and signs, 297–299
    syndromes, 296–297
    valuation, 299
Odontoid fracture
    C-2, 174
Odor
    from vascular support garments, 656
    See also Smell
Odor identification test, 283
OEA
    See Operations, Evaluations, and Analysis Officer (OEA)
Office of The Surgeon General (OTSG), 20, 22–23, 80
    Army Patient Administration Division, 27
    Reconditioning Division, 22
OH
    See Orthostatic hypotension (OH)
Ohm's Law, 451
Olfactometer, 284
Olfactory evaluation, 283–285
Olfactory evoked potentials, 284
Olfactory hallucination, 283
Olfactory nerve, 282–285
    anatomy and function, 282–283
Olfactory neuropathies, 282–285
    electrodiagnosis, 284–285
    evaluation, 283–284
    imaging, 285
    incidence, 282
    management, 285
    prognosis, 285
    symptoms and signs, 283
Olfactory recognition
    diminished, 283
Olfactory threshold tests, 284
Omeprazole, 754
Omniderm, 594
One repetition maximal lift, 806
One repetition maximum (1RM), 802, 812
Onion skin pattern of Dejerine, 169
Onset of blood lactate accumulation (OBLA), 789
Open axillary abduction splint
    See Airplane splints
Open circular amputation, 45–46, 83, 134
    advantages, 94
    disadvantages, 95
    surgical technique, 94
Open flap technique
    for limb revision, 95
Operation Job Match, 855
Operations, Evaluations, and Analysis Officer (OEA), 877
Ophthalmic nerve, 300
Ophthalmoplegia, 295–296
Opioid antagonists
    for traumatic brain injury, 216–217, 222
Opioids
    and traumatic brain injury, 218
Opponens hand splints, 705–707
Opsite, 593–594
Optic atrophy, 287
Optic chiasm, 288
Optic nerve, 285–291
    anatomy and function, 285–286
Optic neuropathies, 286–290
    evaluation, 288–290
    incidence, 286–287
    management, 291
    prognosis, 291
    symptoms and signs, 287–289
    syndromes, 287

Optic radiations, 286, 289
Optic tract, 286, 289
Optokinetic reflex, 294
Oral contractures
  management, 638
Oral phase
  of swallowing, 327
"O" ring
  in upper extremity prostheses, 55, 58
Orthopedic felt, 668
Orthopedic mechanics
  See Prosthetists
Orthopedics
  in combat theater, 8
  history, 830–831
  Persian Gulf War, 27
  role, 15
  Vietnam War, 24
Orthopedic trauma
  immobilization for, 743
Orthoplast, 617
Orthoses, 703–740
  algorithm for use of, 612
  for burn patients, 596, 600, 611–618, 629–630, 637–643
  commonly used materials, 617
  compression inserts, 650, 664–674
  contact dermatitis caused by, 668–669
  lower extremity, 704, 715–737
  for nerve injuries, 12–13
  overlays, 664–674
  for peripheral nerve injuries, 471–472, 492, 495
  postgraft immobilization, 629–631
  for shin splints, 396
  for skeletal insufficiency, 733–737
  upper extremity, 704–715
  See also specific type of orthosis
Orthostasis
  immobility-related, 748–749, 765
Orthostatic hypotension (OH)
  in spinal cord injured patients, 175, 177, 184
Orthotists, 832
Osteomyelitis, 134
  in spinal cord injured patients, 187–188
Osteomyoplasty, 95–96
Osteoperiosteal bone flaps, 95–96
Osteoporosis
  immobilization, 747–748, 750
  in spinal cord injured patients, 189
Otoform K, 667, 670
Otoscopic examination, 321
Ototoxic drugs, 323
OTSG
  See Office of The Surgeon General (OTSG)
Otto Bock 1D10 Dynamic Foot, 120
Otto Bock hands, 69–70
Outpatient care
  for burns, 583–584, 636
  transition from inpatient care to, 858–859
  for traumatic brain injury, 227–228
Overhead walkers, 604–605
Overlays
  for orthoses, 664–674
Overload, 472, 807
  musculoskeletal, 357, 359–360
  physiological response of bone to, 817
  progression, 807
Overstretching
  complications, 466
Overt Aggression Scale, 223
Overtraining, 809
Overweight, 793
  definition, 793, 797
Overwork injury
  clarification, 467–468
  foot and ankle, 531–532
Overwork weakness
  definition, 467
Oxcarbazepine, 222
Oxford technique, 469
Oxidative capacity
  of muscle, 801
Oxidative phosphorylation, 786–787
Oxotremorine, 218
Oxycodone, 622
Oxygen (O_2)
  partial pressure (PO_2), 789, 791
  utilization, 788
Oxygen consumption (VO_2), 787, 789
  and cardiac output, 790
  See also Volume of maximum oxygen consumption (VO_2max)
Oxygen content
  arterial, 801
Oxygen transport system
  factors that limit, 792, 800–801
Oyster shell orthoses, 655

P

Paced Auditory Serial Addition Test, 243
PAFOs
  See Plastic ankle-foot orthoses (PAFOs)
Pain
  acute, 621
  back, 11, 378–389, 743, 857
  burn, 621–622
  causalgia-associated, 482
  chronic, 621
  deafferentation, 475
  dysesthetic, 475
  facial, 304, 308
  heel, 536–537
  nerve trunk, 475
  neuropathic, 475
  patellofemoral, 394–395
  posterior element, 384–385
  at residual limb/prosthesis interface, 142–143
  retroauricular, 312
  in spinal cord injured patients, 191–192
  throat, 332–333
  See also Phantom limb pain; Residual limb pain
Pain clinics
  for phantom limb pain, 147–148
Pain management
  for burn patients, 599–600, 621–623, 625–626
  for causalgia, 489–492, 495
  for lower extremity amputation, 88, 125, 134, 147
  for musculoskeletal disorders, 357–358
  for peripheral nerve injuries, 474–479
  for spinal cord injured patients, 191–192
  See also specific type of drug or treatment
Pain syndromes
  sympathetically maintained, 428, 481
Palatal arch
  evaluation, 327
Palatal reflex, 327

Palate
  defective elevation, 332
  examination, 333
Palmar stimulation
  for carpal tunnel syndrome, 512
Palm orthosis, 640–641, 667
Pamidronate, 750
Panama, 23
Papaverine hydrochloride, 219
Papilledema, 287
Parallel bars
  and amputees, 103–104, 148
  and spinal cord injuries, 177–178
Paralytic ileus
  in burn patient, 602
Paraosteoarthropathy
  See Heterotopic ossification (HO)
Paraplegia
  See Spinal cord injury (SCI)
Paraplegic ambulation, 177–178, 731
  energy expenditure, 731
Parathyroid hormone levels
  in immobilized patients, 750
Paratrigeminal syndrome, 303
Parenteral nutrition support
  for burn patients, 688
Parinaud's syndrome, 297
Parkinsonism
  after traumatic brain injury, 220, 222
Parkland shoe, 615
Parosmia, 283
Paroxetine, 215–216, 220
Pars interarticularis injury, 385–386
Pars stress reaction, 385
Partial anterior interosseous syndrome, 509
Partial calcanectomy, 87
Partial foot amputations, 85–93
  joint contractures, 88
  long-term follow-up, 92
  prosthetic fitting and training, 89–92
  rehabilitation, 87–89
  surgical technique, 85–87
  See also specific type of amputation
Partial pressure of carbon dioxide (PCO_2), 789
  response to maximal exercise, 791
Partial pressure of oxygen (PO_2), 789
  response to maximal exercise, 791
Partner-resisted exercises, 812
PA Section
  See Physician Assistant (PA) Section
Passive prehension orthoses, 709
Passive range-of-motion (PROM)
  for burn patients, 605–606, 616–618
  contraindications, 603
  definition, 602
Passpointing, 321
Patch test
  for allergic contact dermatitis, 139
Patellar tendon bearing, total contact socket (PTB-TCS), 107, 139
  hard, 108
  soft, 107
  supracondylar, 108–109
Patellar tendon-bearing orthosis, 735–736
Patellar tendon-bearing prosthesis
  alignment check using, 103–104
Patellofemoral pain syndrome, 394–395
Patient
  role in interdisciplinary team, 833
Patient age
  See Age; Children; Elderly
Patient controlled analgesia (PCA), 88, 125
Patient education
  after burn injury, 601–602
  after spinal cord injury, 175–180, 186–187
  after traumatic brain injury, 231, 245
  in hand rehabilitation, 38
  and hearing impairments, 323–325
  for lower extremity amputees, 84, 101–102, 133, 138
  for musculoskeletal injuries, 382–383
  for upper extremity amputees, 52, 65, 68–69
Patient Evaluation Conference System, 224
Patient history
  in disability compensation evaluation, 868–869
Patten bottom
  orthoses with, 734–735
Pattern recognition system
  using phantom limb sensation, 66
Pavlov ratio, 165
PCA
  See Patient controlled analgesia (PCA)
PDB
  See Physical Disability Branch (PDB)
PEB
  See Physical Evaluation Board (PEB)
PEBLO
  See Physical Evaluation Board Liaison Officer (PEBLO)
Pectoralis minor syndrome, 501
PEEP
  See Positive end-expiratory pressure (PEEP)
Pelvic bands
  with knee-ankle-foot orthoses, 730–731
Pelvic fractures
  lumbosacral plexopathy caused by, 523–524
Pelvic tilts, 384
Pemoline, 214–215
Penetrating nerve injuries, 446–448
  case studies, 491–496
  classification, 429
  clinical considerations, 447–448
  pathophysiology, 446–447
  prognosis, 447–448
Pensions
  See Physical disability system
Pentazocine, 503
Pentoxifylline, 219
Peptic ulcer disease, 753–755
Percy Jones General Hospital, Michigan, 35, 80
Pergolide, 214–215
Perineurium, 427
  and traction-related neuropathies, 442
  transection, 430–431
Peripheral nerve injuries, 419–574
  in burn patients, 689–690
  classification, 429–431
  cold-induced, 443–446
  compression, 436–439
  electrical muscle stimulation, 481
  electrical stimulation intervention, 479–481
  electrodiagnosis, 448–465
  end organ changes following, 431–436
  epidemiology, 420–422
  foot and ankle, 531–545
  hyperesthetic desensitization therapy, 472–473
  ischemic, 439–440
  localization, 461–463

  lower extremity, 522–531
  mechanisms, 436–448
  orthotic management, 471–472, 492, 495
  pain management, 474–479
  penetrating, 446–448, 491–493
  Persian Gulf War, 11–14, 420, 422, 446, 525
  pharmacological intervention, 475–479
  prognosis, 459–462
  range-of-motion maintenance, 465–467
  rehabilitative management, 465–481
  reinnervation following, 431–432, 434, 436, 460, 463
  secondary injury prevention, 435, 465
  sensory reeducation, 473–474
  severity determination, 459–462
  in spinal cord injured patients, 192–193
  strength maintenance and improvement, 467–470
  stretch, 440–443
  surgical repair postponement, 465
  upper extremity, 496–522
  Vietnam War, 447, 497
  during World Wars, 420–422, 447
  See also Causalgia; *specific injury*
Peripheral nerves
  anatomy, 422–423, 426–427
  axonal transport, 425–426
  causalgia-associated changes in, 484
  degeneration, 431–434
  effect of immobility on, 749
  fascicular composition, 426–427, 435, 438–439, 442
  impulse propagation, 423–425
  neuroanatomy, 422–423
  physiology, 423–426, 448–450
  regeneration, 431–432, 434, 436, 460, 463
  vascular system, 427–428
Peroneal nerve, 527–528
  deep, 538
  functional electrical stimulation, 725–726
  superficial, 540
Peroneal neuropathy, 527–531
  anatomic considerations, 527–528
  in burn patients, 689
  case scenario, 493–496
  clinical presentation, 529
  dislocation-associated, 440–441
  electrodiagnosis, 529–530
  etiology, 528–529
  fracture-associated, 441–442
  superficial, 540–542
  treatment, 530–531
PERSCOM
  See Total Army Personnel Command (PERSCOM)
Pershing, John J., 11
Persian Gulf War, 23
  amputations, 12–15, 46–47, 80
  Army Physical Therapy Registry, 27–28
  brain injuries, 15
  burn injuries, 14–15
  causalgia, 482, 491
  disability case statistics, 879–880, 883
  functional impairments, 15
  hand rehabilitation, 38
  immobility complications, 742, 745, 756
  musculoskeletal injuries, 11–13
  nerve injuries, 11–14, 38, 80, 420, 422, 446, 525
  physical therapy, 25–28
  rehabilitation services, 10–16
  rehabilitation teams, 7
  wrist-hand orthoses use, 709
Personality
  after traumatic brain injury, 217, 223, 238–239
  premorbid, 195–196
Personnel Management Officers (PMOs), 865, 877
PFTs
  See Pulmonary function tests (PFTs)
Phalangization, 36–37
Phalen's test, 511
Phantom limb
  pattern recognition system using, 66
Phantom limb pain, 14, 143–147, 475
  definition, 143
  differential diagnosis, 148
  duration and frequency, 144
  incidence, 143–144
  in lower extremity amputees, 88
  pathophysiology, 126, 144–145
  prevention, 147
  treatment, 145–148
  in upper extremity amputees, 61
  See also Residual limb pain
Phantom limb sensation
  after lower extremity amputation, 126, 143
  classification, 143
  definition, 143
  explanation, 126, 143
  fading, 143
  management, 126, 143
  telescoping, 88, 143
Pharmacologic therapy
  and alcohol use, 240
  auditory complications, 323
  for burn patients, 599–600
  debilitating side effects, 851
  for immobilization osteoporosis, 748
  ocular complications, 296
  for peripheral nerve injuries, 475–479
  for phantom limb pain, 126, 145–146, 148
  for traumatic brain injury, 214–222, 232–233
  See also specific drug or drug class
Pharyngeal phase
  of swallowing, 328
Pharyngeal reflex, 327
Phenobarbital, 221
Phenoxybenzamine, 490
Phenytoin, 477, 490
  for cranial neuropathies, 308
  for traumatic brain injury, 216, 221
Phosphagen system, 787–788
Phosphorylation
  oxidative, 786–787
Photographs
  in burn wound management, 599, 655
Photoreceptors, 285–286
Phthalazinol, 217
Physiatrist
  definition, 4, 6, 229, 832
  duties beyond physical rehabilitation, 8
  role in interdisciplinary team, 832
  role in surgical decision process, 83, 96
  role in traumatic brain injury care, 226, 229
Physiatry
  history, 4–6, 831
  See also Interdisciplinary team
Physical activity
  as risk factor for injury, 817
Physical capacity for exercise, 799–803
  aerobic, 803–806

  and age, 804–805
  and body composition, 794–796
  and gender, 803–804
  of military populations, 803–806
  and occupation, 804
Physical Disability Branch (PDB), 865, 873
Physical disability systems, 863–885
  Air Force, 884
  Army, 865–883
  history, 864–865
  Navy, 883–884
  statistics, 879–883
  triservice comparison statistics, 883
Physical Evaluation Board (PEB), 71, 150, 250, 865–866, 872–873
  caseload statistics, 879–883
  formal hearing process, 874–876
  location, 867
  versus Medical Evaluation Board (MEB), 872
  Navy, 883
  and preexisting conditions, 876
  presumptions applied to, 876–877
  process, 873–877
  USAPDA review, 876–879
Physical Evaluation Board Liaison Officer (PEBLO), 868, 872–874
Physical examination
  for disability compensation evaluation, 869
Physical fitness, 781–827
  and body composition, 792–799
  definition, 782
  and disability compensation evaluation, 873
  and musculoskeletal disorders, 360
  as risk factor for injury, 817
  See also Physical capacity for exercise
Physical fitness standards
  and age, 804–805
  DoD directive, 782, 796–799
  and gender, 803–804
  and training programs, 811
Physical medicine and rehabilitation (PMR)
  in combat theater, 8–9
  at medical centers, 9–16
  overview, 4, 6–8, 229
Physical profile
  in physical disability system, 867–868
Physical therapists
  Army, 7, 20, 24–25, 28–29
  in civilian practice, 25
  deployed to third world countries, 28
  and early ambulation, 84
  education, 25
  role in interdisciplinary team, 831–832
  shortage, 28
  Vietnam War, 23–24
Physical therapy
  for above-knee amputees, 133
  Army, 16, 20–28
  in evolving doctrine, 28–29
  history, 830–831
  for lower extremity amputees, 103–104, 106
  for mild traumatic brain injury, 231
  for multiple amputees, 149
  Persian Gulf War, 25–28
  for spinal cord injured patients, 175
  Vietnam War, 23–24
  in wartime environment, 19–30
Physical therapy technicians, 830
Physical tolerance
  and vocational assessment, 851
Physical training, 782, 807–811
  adaptive response to, 807, 809–810
  cardiovascular disease associated with, 818
  definition, 807
  duration, 807, 810, 812–813
  frequency, 807, 810–813
  heart rate-based, 790
  intensity, 807, 810, 812–813
  job-specific, 808, 814
  length, 810–811
  medical problems associated with, 814–820
  mode, 810–811, 813
  musculoskeletal injuries associated with, 814–817
  principles, 807–809
  specificity, 808
  and weight control programs, 793–794
  See also Exercise; *specific type of exercise*
Physical training programs, 811–814
  components, 810–811
  initiation, 811
  integrated, 813–814
  objectives, 811
  as risk factor for injury, 816–817
Physician Assistant (PA) Section, 23
Physicians
  role in Army physical disability system, 867–868
  role in interdisciplinary team, 831
Physiotherapy aides
  World War I, 21
Physostigmine, 218
Pigskin, 582
Pin fixation
  for limb length preservation, 95
PIP joint
  See Proximal interphalangeal (PIP) joint
Piracetam, 219, 221
Piriformis syndrome, 525
Pirogoff hindfoot amputation, 86–87
Pistoning
  of ankle-foot orthoses, 722–723
Plantar digital nerve, 543
Plantar fasciitis, 400–401
Plantar flexion (posterior) stops
  on ankle-foot orthoses, 715, 717–718, 722–723
Plantar nerve
  lateral, 532–533, 536–537
  medial, 532–533, 537–538, 543
Plastazote, 670
Plaster of Paris
  in immediate postoperative prostheses, 97–100
  socket design with, 121, 130
Plaster orthoses, 617
Plastic ankle-foot orthoses (PAFOs), 720–723
Plastic deformation
  factors influencing, 465–466
Plastic materials
  in sockets, 130
Plastic stretch, 465
Plastic surgery
  for burn injuries, 690–693
Plug fit socket, 129
Plumb line
  alignment check using, 103–104
PMOs
  See Personnel management officers (PMOs)
PMR

  See Physical medicine and rehabilitation (PMR)
Pneumatic compression
  intermittent, 471
  for lower extremity amputations, 87, 124, 127
Pneumonia
  in spinal cord injured patients, 182
PNF
  See Proprioceptive neuromuscular facilitation (PNF)
Polio epidemics, 6
Pollicization
  of remaining fingers, 36–37
Polycushion, 668–670
Polyflex, 617
Polyform, 617
Polymyxin, 591
Polyurethane membranes, 593–594
Polyurethane pads
  in immediate postoperative prostheses, 96–97
Porcine skin heterograft, 593
Positioning
  during acute phase of burn care, 607–611
  antigravity, 604, 607–610, 628, 743, 746
  during immobilization phase of burn care, 628–629
  for pressure sore prevention, 757
  during wound maturation phase of burn care, 637
Positive end-expiratory pressure (PEEP), 599
Positive sharp waves, 454–456, 494
  quantification, 455
Postconcussive symptoms, 230–231
  pharmacologic therapy, 232–233
Postconcussive syndromes
  neurodiagnostic evaluation, 232
Posterior cord syndrome, 170
Posterior element pain
  acute, 384–385
Posterior flap method
  of lower extremity amputation, 95
Posterior interosseous nerve, 503–504
Posterior interosseous nerve entrapment, 503–505
Posterior stops
  on ankle-foot orthoses, 715, 717–718, 722–723
Posterior tibial nerve, 532
  See also Tarsal tunnel syndrome
Postpolio syndrome, 467
Posttraumatic amnesia (PTA)
  and family outcome, 253
Posttraumatic stress disorder (PTSD)
  in burn patients, 631, 683–684
  and vocational rehabilitation, 858
Posture
  and musculoskeletal injuries, 362–364
Posturography, 321
Postvoid residual volume
  determination, 752
Potassium depletion
  immobilization-induced, 750
Potential reserve, 743
Povidone-iodine, 582, 597
Power sources
  for hand orthoses, 705, 708–709
Powers ratio, 173
Power tests
  anaerobic, 801–802
Pramiracetam, 219
Prazosin, 220
Prednisone, 357, 490
Preexisting conditions
  and disability compensation review, 876
Pregnancy
  in spinal cord injured patients, 195
Prentke Romich Light Talker, 620–621
Prentke Romich Touch Talker, 620–621
Presbycusis, 319
Pressure intolerant areas
  in sockets, 107
Pressure relieving devices, 757
Pressure sores, 162, 181, 186, 190, 755–758
  causes, 186, 755
  classification, 187, 757–758
  distribution, 755–756
  identification of patients at risk for, 756–757
  incidence, 187, 756
  prevention, 186, 756–757
  treatment, 187, 758
Pressure tolerant areas
  in sockets, 107
Pressure wrap
  bulky, 629–630
Prilosec
  See Omeprazole
Primary irritant dermatitis
  on residual limb, 139
Primidone, 221
Progression
  overload, 807
Projectile-related injuries
  See Bullets; Penetrating nerve injuries; Shell fragment wounds
Prolonged stretch
  for burn patients, 603, 636
  definition, 603
  with diathermy, 746
PROM
  See Passive range-of-motion (PROM)
Pronator teres muscle, 506–507
Pronator teres syndrome, 506–508
Prone positioning
  anticontracture, 608, 746
Proportional control systems
  for myoelectric prostheses, 65–67
Propranolol, 217, 220, 490
Proprioception changes
  burn injury-associated, 633
Proprioceptive neuromuscular facilitation (PNF), 359
Proprioceptive training
  for musculoskeletal injuries, 393, 398
Prostheses
  bypass, 126
  and early ambulation, 13
  exoskeleton, 132
  fabrication, 80
  facial, 655
  for multiple amputees, 148–149
  shower, 133
  stubbie, 133, 148–149
  temporary, 12–14, 84, 88
  World War II technology, 35
  See also Myoelectric prostheses; *specific type of prosthesis*
Prosthetic foam, 670
Prosthetic sockets
  See Sockets
Prosthetists, 80, 133, 832
Protein
  and burn injuries, 687–688
  status evaluation, 689
  synthesis, 750

Prothrombin time (PT), 763
Proximal interphalangeal (PIP) joint
  amputation at, 37
Proximity
  principle of, 623
Pruritus
  after burn injury, 633
Pseudo-anterior interosseous syndrome, 508
Pseudo-Graefe's sign, 299
Pseudo-Graefe's syndrome, 297
Pseudo-obstruction
  in spinal cord injured patients, 185
Psychiatrists
  role on medical evaluation board, 868
Psychological disturbances
  with amputation, 10
  immobility-related, 749
  treatment, 16
Psychological shock, 624
Psychological support
  after brain injury, 227–228
  for burn patients, 596, 623–626, 631, 633, 683–684
  for immobilized patients, 750
  in interdisciplinary team management, 832
  for lower extremity amputees, 84, 88, 101, 125, 133–134
  for multiple amputees, 149
  for phantom limb pain, 147–148
  and physical recovery, 846
  for spinal cord injured patients, 180, 195–196
  for upper extremity amputees, 47, 52
  See also Emotional counseling; Psychotherapy
Psychologists
  role in interdisciplinary team, 832
Psychosocial outcome
  determinants of, 846–847
  of traumatic brain injury, 238–241
Psychostimulants
  for traumatic brain injury, 214–215, 219
  See also specific drug
Psychotherapy
  for brain injured patients, 245–246
  for burn pain management, 623
  for phantom limb pain, 147–148
Psyllium hydrophilic mucilloid fiber, 753
PT
  See Prothrombin time (PT)
PTA
  See Posttraumatic amnesia (PTA)
PTB-TCS
  See Patellar tendon bearing, total contact socket (PTB-TCS)
Pterygoid reflex
  electrodiagnostic evaluation, 305–306
Ptosis, 297, 299
PTs
  See Physical therapists
PTSD
  See Posttraumatic stress disorder (PTSD)
Pubis stress fractures, 397
Public Health Service
  physical therapy services, 25
Public Law No. 101-336
  See Americans with Disabilities Act
Pulmonary angiography, 760–761
Pulmonary complications
  of immobilization, 758–762
  of spinal cord injury, 181–182
  See also specific complication
Pulmonary compromise
  after spinal cord injury, 181
  signs of, 583
Pulmonary edema
  in burn patients, 584–585
  high-altitude, 820
Pulmonary embolism, 183, 760–762
  versus deep venous thrombosis, 763
  diagnosis, 760–762
  prevention, 762
  treatment, 762
Pulmonary function
  during exercise, 788–789, 801
Pulmonary function tests (PFTs), 758–759
Pulmonary management
  of burn patients, 584–585, 598–599
  of immobilized patients, 758–762
Punctate hemorrhages, 209
Pupil
  muscles regulating, 294
  myotonic, 297
Pupillary light reflex, 288–290, 296
  innervation, 294
Pushoff
  with metal ankle-foot orthoses, 718–720
  with plastic ankle-foot orthoses, 722–723
Pylon
  in immediate postoperative prostheses, 98–100, 135
Pyridoxine, 513

Q

Quadrilateral sockets, 129
Quadriplegia
  See Spinal cord injury (SCI)
Quadruple amputation, 149
Quality of life
  for spinal cord injured patients, 195
  for traumatic brain injured patients, 224
Quisqualate, 212

R

Racial groups
  counseling concerns, 849
Radial amputations
  definition, 40
  prostheses, 39–42
Radial nerve, 496, 502
Radial neuropathy, 438–439, 502–505
  anatomic considerations, 502–503
  case scenario, 491–493
  clinical presentation, 503
  compression, 505
  electrodiagnosis, 503
  etiology, 503
  fracture-associated, 440–441
  orthotics, 640–641, 705–706
  in spinal cord injured patients, 192
  treatment, 503–504
Radiculopathy
  cervical, 363–366
  lumbar, 379–381
Radiography
  in musculoskeletal injuries, 381, 385–386
  in residual limb problems, 140–143
  in spinal cord injury, 163, 188
  in tarsal tunnel syndrome, 535
Raeder's syndrome, 303
Ramsay-Hunt syndrome, 311–312

Rancho Los Amigos Scale, 224
Range of motion
  definition, 602–603
  and hand rehabilitation, 38
  and peripheral nerve injuries, 465–467
Range-of-motion exercises
  for accessory neuropathies, 338
  for brain injured patients, 226
  for burn patients, 596–597, 602–606, 628
  early initiation, 9
  for edema control, 470
  for immobility complications, 746
  for lower extremity amputees, 84, 101–103, 135
  for musculoskeletal injuries, 359, 362, 372, 376–378
  for peroneal neuropathy, 531
  for phantom limb pain, 147–148
  for spinal cord injured patients, 189
  for upper extremity amputees, 47
Ranitidine, 754
Rash
  heat, 819
Ratchet splint, 709
Rating by analogy, 873–874
Ray amputations
  prosthetic fitting and training, 89
  surgical technique, 86
Raynaud's phenomenon, 444
Ray resection
  for finger amputations, 37–38
Reach
  orthotic substitution for, 704–706, 710–713
Reachers, 179, 679
Reading
  adaptive devices, 621
Reamputation (revision), 83, 134, 148
  surgical technique, 94–95
Reappraisal, 847
Reciprocating gait orthoses (RGOs), 732
Reconstruction aides
  World War I, 20–21
Reconstructive surgery
  for burn patients, 690–693
Recovery principle
  in training, 807
Recreational adaptive aids, 10, 41–42, 52–54, 70–71, 149, 682–683
Recreational therapists
  role in interdisciplinary team, 832
Recreational therapy
  for above-knee amputees, 133
  for brain injured patients, 227
  for burn patients, 636–637, 682–683
  definition, 832
  for spinal cord injured patients, 179–180, 195
  See also Exercise; Sports; *specific activity*
Rectus muscles
  innervation, 292–293
Redundancy
  after traumatic brain injury, 212
Reed College, Oregon, 20
Referral questions
  for neurobehavioral assessment, 242–243
Reflex arc, 785
Reflex detrusor contractions
  in spinal cord injured patients, 193
Reflexes
  facial nerve, 312–314
  glossopharyngeal nerve, 327
  hyperactive, 190–191
  laryngeal nerves, 333
  segmental, 171–172
  trigeminal nerve, 302–307
  vagus nerve, 331
  See also specific reflex
Reflex hyperventilation, 789
Reflex sympathetic dystrophy (RSD), 141–142, 148, 749
Regional blockade
  for causalgia, 489–490
Rehabilitation nurse
  role in interdisciplinary team, 832–833
Rehabilitation services
  Persian Gulf War, 7, 10–16
  Vietnam War, 7–10
Rehabilitation team
  See Interdisciplinary team
Reichert's syndrome, 326–327
Reinnervation
  after peripheral nerve injury, 431–432, 434, 436
  after traumatic brain injury, 212
  electrodiagnostic monitoring, 460, 463–464, 468
  and functional recovery, 464–465
Reiter, Reinhold, 65
Relaxation techniques
  for burn pain management, 600, 623, 625–626
  for phantom limb pain, 147
Remodeling phase, 355
Remotivation, 847
Renal calculi
  in spinal cord injured patients, 194
Renal failure
  in spinal cord injured patients, 193
Reparative phase, 354–355
Reserpine, 490
Residual limb
  definition, 43
  edema, 134, 138
  electrical stimulation, 67
  and energy expenditure for walking, 93
  hygiene concerns, 84, 107–108, 134, 136
  interface with lower extremity prostheses, 89, 96–97, 104, 107, 129, 131, 136–137, 142–143
  lengthening, 96
  in lower extremity amputees, 134, 136
  in multiple amputees, 148–149
  plaster cast impression, 121
  topographic measurement, 121
  in upper extremity amputees, 61
  very short below elbow, 44, 53
  vibration/percussion, 147
  weight bearing, 91–92, 98–99, 102–103
Residual limb pain
  causes, 139–143
  differential diagnosis, 142, 148
  postoperative management, 88, 125, 134, 147
  referring, 142, 148
  See also Phantom limb pain
Residual limb pain syndrome, 14
Residual limb wrapping
  See Stump wrapping
Resin
  in above-knee prostheses, 130
  in foot prostheses, 90
Resistance
  negative, 813
  variable, 813
Resistance detraining, 808

Resistance training, 810–813
  adaptation of aerobic training modes to, 812
  adaptive response to, 809–810
  cardiovascular response to, 809–810
  equipment, 812
  for lower extremity amputees, 103
  for muscle mass buildup, 793–794
  for musculoskeletal injuries, 359–360
  for peripheral neuropathy, 467
  specificity, 808
  in weight control programs, 793
  See also Isotonic exercises; Strength training
Respirators
  for burn patients, 599
Respiratory failure
  after spinal cord injury, 181
  signs of, 583
Respiratory management
  of burn patients, 584–585, 598–599
  of immobilized patients, 758–762
Respiratory quotient (RQ), 686
Respiratory tract infection
  in immobilized patients, 759
Reston foam, 668
Restructuring, 847
Retina, 286, 288
Retired List
  history, 864–865
  Temporary Disability (TDRL), 865, 873–874, 876, 883
Retirement
  See Physical disability system
Retroauricular pain, 312
Return to duty
  of amputees, 150
  of brain injured patients, 246–250
  of burn patients, 637, 683–685
  importance, 16
  of lower extremity amputees, 106
  of multiple amputees, 149
  and musculoskeletal disorders, 360
  transition to, 858–859
  of upper extremity amputees, 70–73
  during Vietnam War, 9, 23–24, 72, 150
  See also Vocational assessment; Vocational rehabilitation
Revision
  See Reamputation (revision)
RGOs
  See Reciprocating gait orthoses (RGOs)
Rhabdomyolysis
  exertional, 818
  in spinal cord injured patients, 193
Rheobase, 449
Rheumatologic disorders
  in amputees, 142
Rhodopsin, 285–286
Rigidity, 208
Ring fracture
  C-1, 173–174
  C-2, 174
Rinne's test, 320–321
Ritalin, 219
1RM
  See One repetition maximum (1RM)
Rocker bottom
  orthoses with, 734–735
Rocker soles
  in shoes, 90, 114
Rods, 285–286
Roentgenography
  in anterior tarsal tunnel syndrome, 539
  in causalgia, 486–487
  in pulmonary embolism, 760–761
Roger's sign, 304
ROHO bed, 611, 757
Rolyan Silicone Elastomer, 667
Romania, 28
Romberg test, 321
Room temperature
  for burn patients, 599–600
Roosevelt, Franklin D., 5, 21
Rotating beds
  for spinal cord injured patients, 182, 186
Rotator cuff injuries, 367–370
  in spinal cord injured patients, 189
Rotobed, 631
Roto-Rest Traumabed, 182, 186
RQ
  See Respiratory quotient (RQ)
RSD
  See Reflex sympathetic dystrophy (RSD)
Rucksack palsy, 497–498
Rule of 9s, 580
Rule of the Palm, 579–580
Rusk, Howard A., 5, 831
Russia, 28

S

Sabolich foot, 120
Saccadic deficits, 299
Saccadic system, 294
SACH foot
  See Solid-ankle cushion heel (SACH) foot
Sacral plexus, 522–523
Saddle harness
  for above-elbow prostheses, 57–58
SAFE (stationary attachment, flexible endoskeleton) prosthetic foot, 92, 114–115, 120
Sagittal flap method
  of lower extremity amputation, 95, 124
Saliva
  hypersecretion, 333
  pooling, 312
Salivary flow meter, 312
Salivation
  loss, 312
  measurement, 328
Salmon calcitonin, 748
Saltatory conduction, 449
Salt depletion, 819
Sanderson, Marquerite, 20–21
Saphenous nerve, 526
Sarcomeres, 783
Saturday night palsy, 192, 503
Saudi Arabia
  See Persian Gulf War
Scald burns, 577
Scalp donor
  use and healing, 594
Scapular retractors/stabilizers, 368
  strengthening exercises, 363
Scar(s)
  in burn patients, 643–674
  hypertrophic, 643–645, 655–674
  location on residual limb, 136
  skin grafts, 644

Scar compression, 655–674
  ankle, 675
  arm, 673
  elbow, 672–673
  fingers, 671
  foot, 676
  hand, 672
  hip, 675
  knee, 675
  lower extremity, 675–676
  shoulder, 673
  thumb, 671–672
  trunk, 674
  upper extremity, 671–673
  wrist, 672
Scarpa's triangle, 129, 139
Schirmer lacrimation test, 312
Schmidt's syndrome, 332
Schwann cells, 422–423
  denervation-related changes in, 433
  and reinnervation, 434
SCI
  See Spinal cord injury (SCI)
Sciatic nerve, 524, 528
Sciatic neuropathy, 438, 524–526
  anatomic considerations, 524–525
  clinical presentation, 525
  electrodiagnosis, 525–526
  etiology, 525
  Persian Gulf War, 525
SCL-90
  See Symptom Checklist-90 (SCL-90)
Scoop type prostheses, 42–43
Scott-Craig orthoses, 729, 732
Seat-belt type injuries
  spinal, 175
Seattle Ankle, 116
Seattle Foot, 115–116, 120
Seattle Lightfoot, 92, 115–116, 120
Seattle orthosis, 721
Second General Hospital, Germany, 11
Sedation
  of burn patients, 622, 626
  for phantom limb pain, 146
Seddon's classification
  of peripheral nerve injuries, 429
Segmental dysfunction
  lumbar, 379
Segmental hypermobility
  lumbar, 380, 383
Seizures
  posttraumatic, 221–222
Seizure threshold
  effects of alcohol on, 240
Selective serotonin reuptake inhibitors (SSRIs)
  for traumatic brain injury, 216
Self-care
  See Activities of daily living (ADL); *specific activity*
Self-Directed Search, 854
Self-feeding
  adaptive devices, 619–620, 676–677
Self-management skills
  assessment, 855
Sensory abnormalities
  causalgia-associated, 482
Sensory aphasia, 320
Sensory deprivation
  in immobilized patients, 748–750
Sensory feedback
  in lower extremity amputees, 85, 93
  with myoelectric prostheses, 67, 69
Sensory fibers
  special, 280–281
Sensory levels
  for neurological evaluation of spinal cord injury, 169
Sensory nerve action potential (SNAP)
  amplitude, 459, 464
  in brachial plexopathies, 500
  in carpal tunnel syndrome, 512
  in compression injuries, 437
  inability to elicit, 460
  measurement, 451
  in thoracic outlet syndrome, 501–502
  in ulnar neuropathy, 518
Sensory recovery
  after peripheral neuropathy, 464–465
  classification, 474–475
Sensory reeducation, 473–474, 492
  discriminative, 474
  protective, 474
Sensory stimulation
  and burn patients, 631
  and comatose patient, 236
  and immobilized patients, 749–750
Sensory Stimulation Assessment Measure, 224
Separated without benefits (SWOB), 876
Separated with severance pay (SWSP), 876
Serial casting, 747
Series elastic component, 785
Serotonergic drugs
  for traumatic brain injury, 215–217, 221
  See also specific drug
Serotonin, 476
Sertraline, 215–217, 220
Servo Pro system, 66–67
Sexual counseling
  after brain injury, 254
  after burn injury, 635
Sexual function
  in burn patients, 634–635
  in spinal cord injured patients, 194–195
  in traumatic brain injured patients, 222
Shanks
  for below-knee prostheses, 112–113
  endoskeletal, 113
  exoskeletal, 113
  thermoplastic, 109–110
Shear forces
  with knee-ankle-foot orthoses, 727–729
Sheepskin, 668
Sheet grafts, 589, 644
Shell fragment wounds
  brachial plexopathies caused by, 497
  burns secondary to, 581
Shin splints, 395–396
Shock
  burn, 585–587
  psychological, 624
  spinal, 172
Shoes
  for ankle-foot orthoses, 718
  for burn patients, 615–616, 641–643
  inserts, 89–90
  as risk factor for injury, 817
  rocker soles, 90, 114
  toe fillers, 89

Shoulder
  contracture prevention, 47
  instability, 370–372
  positioning for burn injuries, 609–610
  scar compression, 673
Shoulder bypass suspension hoops, 714–715
Shoulder cap design, 714
Shoulder continuous passive motion machine, 618
Shoulder disarticulation, 43–45
  prostheses, 45, 59, 67
  surgical procedures, 46
Shoulder dislocation
  stretch neuropathies associated with, 440–441
  treatment, 370–371
Shoulder disorders, 367–372
  in spinal injured patients, 189
  See also specific disorder
Shoulder flexion assists, 714–715
Shoulder harness-driven flexor hinge orthoses, 708
Shoulder orthoses, 630, 638–639
  functional, 713
  static, 713
  support of, 713–715
Shoulder stabilizers
  orthotic, 713
Shoulder suspension
  for above-knee prostheses, 131
Shower prostheses, 133
Shower trolley method
  of wound cleansing, 598–599
Shrinker socks, 104–105
Sickness Impact Profile, 253
Side-lying positioning
  anticontracture, 608, 746
SIDS
  See Sudden infant death syndrome (SIDS)
SII
  See Strong Interest Inventory (SII)
Silastic, 670
Silesian belt, 131, 133
Silicone
  in above-knee prostheses, 132
  in foot prostheses, 90
  in orthotic inserts and overlays, 670
  suction sockets, 112, 131
Silicone finger trough, 615
Silver nitrate, 582
Silver sulfadiazine, 582
Singer procedure
  for lower extremity amputation, 96
Single-axis foot prosthesis, 113–114
Single-axis knee prostheses, 128
Single-control system
  for below-elbow prostheses, 55
Single photon emission computed tomography (SPECT)
  in musculoskeletal injuries, 381, 386
  in traumatic brain injury, 225, 231
Sinus arrest
  in spinal cord injured patients, 182–183
Sitting
  anticontracture position, 610
  by spinal cord injured patients, 175–177
Situational assessment
  in vocational rehabilitation, 856
Sjögren's syndrome, 303
Skeletal insufficiency
  orthoses for, 733–737
  See also Dislocations; Fractures; *specific injury*
Skeletal muscle
  See Muscle(s)
Skew flap method
  of lower extremity amputation, 95
Skiing program
  for amputees, 10, 149
Skin
  artificial, 592
  human cadaver, 593
Skin care
  during acute phase of burn injury, 596–607
  during immobilization phase of burn injury, 625–627
  for lower extremity amputations, 85
  during wound maturation phase of burn injury, 632–635
  See also Skin traction management
Skin care pads, 667, 669–670
Skin complications
  in brain injured patients, 226
  causalgia-associated, 482–483
  in immobilized patients, 755–758
  in lower extremity amputees, 134, 136–137
  in spinal cord injured patients, 162, 181, 186–188, 755–758
  in upper extremity amputees, 61
  See also Pressure sores; *specific complication*
Skin desensitization program
  for lower extremity amputations, 88, 125, 141, 148
  for spinal cord injured patients, 191
Skin flaps, 96, 122, 691–693
Skin fold procedure
  for body fat measurement, 797–798
Skin grafts, 589–592
  adherence, 588, 590
  for burn wounds, 587
  cultured epidermal cells, 591–592
  donor sites, 589, 594–595, 644
  dressings, 591–594, 629
  flaps, 96, 122, 691–693
  insensate, 85
  instruments, 587
  meshed, 589–591, 644
  movement, 590
  orthoses, 629–630
  postgraft dressing changes, 626–627
  postoperative immobilization, 627, 629
  for pressure sores, 187, 758
  for reconstruction, 691–693
  scarring, 644
  sheet, 589, 644
  split-thickness, 96, 122, 589
  surgical staples for, 590
  vascular support, 646–649, 655–674
  wound management, 590–591
Skin necrosis
  after amputation, 135
Skin reactions
  allergic, 138–139
Skin tension
  in residual limb, 136
Skin traction management
  errors, 94
  for lower extremity amputations, 83, 94, 100, 124–125
  for upper extremity amputations, 45
Sleep apnea
  obstructive, 182
Sleeve suspension
  for below-knee prostheses, 111
Sling suspensions
  for arm support, 712–713

  shoulder, 713
Slow twitch (type 1) fibers, 784
Smell
  altered sense of, 283–285
  loss of, 282–285
  testing, 283–285
  See also Odor
Smith and Nephew prosthetic foam material, 667
Smoke inhalation injury, 584–585, 598–599, 620
Smooth pursuit system, 294
SNAP
  See Sensory nerve action potential (SNAP)
Snellen charts, 288
Social isolation
  of brain injured patients, 238–239
Social workers
  role in interdisciplinary team, 833
Society for Physical Therapy Physicians, 5
Sock(s)
  for foot prostheses, 116
  for immediate postoperative dressing/prostheses, 97–100, 104–106
  for suction suspension systems, 112, 131
Sockets
  for above-elbow prostheses, 56–57
  for above-knee prostheses, 129–131
  for below-elbow prostheses, 52–53
  for below-knee prostheses, 106–110, 112
  carbon fiber reinforcement of, 91–92
  computer aided design/manufacturing, 120–121, 130
  construction, 109–110
  fabrication, 120–121, 130
  for forequarter amputation prostheses, 59
  hip disarticulation, 132–133
  for humeral neck amputation prostheses, 59
  for knee prostheses, 129–131
  liners, 107–108, 140–141
  loose, 142
  myoelectric electrodes embedded in, 67
  for partial foot prostheses, 89–91
  pressure areas, 107
  and residual limb problems, 61, 107
  Syme's, 91–92
  transfemoral, 124
  transpelvic amputation, 132–133
  See also specific type of socket
Sof-Care alternating pressure mattress, 757
Sof-Kling, 610, 658
Soft tissue
  contractures, 746
  handling during surgical amputation, 122
  musculoskeletal, 354
  retraction, 94
Soft tissue injuries
  cervical, 361–363
  rehabilitation, 8–9
Sole inserts, 642–643
Sole plate
  for ankle-foot orthoses, 718
  rigid, 90
Solid-ankle cushion heel (SACH) foot, 92, 96, 98, 100, 113, 120, 127
Soma, 422–423
Somatosensory evoked potentials
  filter settings, 452
Sorbisan, 594
Sound pressure, 319
Spasticity
  in brain injured patients, 226
  definition, 208
  in spinal cord injured patients, 167, 172, 178, 190–191
SPECT
  See Single photon emission computed tomography (SPECT)
Speech and language therapists
  role in interdisciplinary team, 833
Speech discrimination test, 320
Speech disorders
  after traumatic brain injury, 222
Speech reading, 324
Speech therapy
  for hypoglossal neuropathies, 341–342
  for mild traumatic brain injury, 231
Spenco second skin, 670–673
Spenco skin care pad, 667, 669
Sphincter pupillae muscle
  innervation, 294
Spinal cord changes
  and phantom limb pain, 126, 144–145
Spinal cord compression
  identification, 163
Spinal cord injury (SCI), 14, 161–205
  bilateral knee-foot-ankle orthoses for, 731–732
  causes, 162, 166–167
  classification, 165, 169
  complete, 169, 176–178
  complication management, 181–196
  complication prevention, 165, 181–196
  emergency treatment, 162
  endocrinologic complications, 185
  first and second echelon MTF treatment, 162–163
  gastrointestinal complications, 184–185
  genitourinary complications, 162, 181, 193–194
  head injuries associated with, 195
  imaging studies in, 163–164
  incidence, 162
  incomplete, 169–170, 178
  interdisciplinary team management case study, 841–843
  ligamentous, 173–174
  long-term follow-up, 180–196
  mortality and morbidity, 162, 181
  musculoskeletal complications, 188–189
  neurological complications, 189–193
  neurological evaluation, 167–173
  neurosurgical treatment, 163–165
  nonpenetrating, 165
  orthoses, 703–740
  pathophysiology, 165–167
  patient education, 175–180, 186–187
  penetrating, 165
  prognosis, 170–171
  psychological complications, 195–196
  pulmonary complications, 181–182
  recovery, 167, 175–179, 195
  recreational rehabilitation, 179–180, 195
  rehabilitation, 9, 175–180
  skin complications, 162, 181, 186–188, 755–758
  third echelon MTF treatment, 163
  treatment by level of injury, 173–175
  Vietnam War, 162
  vocational rehabilitation, 179–180, 195, 856
  walking after, 177–178, 731
  wheelchair use after, 175–177
  World War I, 162
  World War II, 162
  zone of injury recovery, 167, 172–173

Spinal Cord Injury Model Systems Program, 162
Spinal cord injury units, 162
  organization, 196
Spinal manipulative therapy, 362
Spinal nerve root entrapment, 381
Spinal nerves, 168
Spinal shock, 172
Spinal trigeminal tract, 169
Spine
  evaluation, 173–175
  instability, 163, 189
  management, 173–175
  stability, 163–164, 387–389
  See also specific part of spine
Spine alignment
  restoration and maintenance, 163
Spine deformities
  in spinal cord injured patients, 189
Spine injuries
  See Spinal cord injury (SCI)
Spine rehabilitation program, 382–385
Spine stabilization exercises, 387–389
Spiral groove area
  injuries and compression in, 502–503
Spirometry
  incentive, 759–760
Splinting, 747
  dynamic, 747
Splints, 358, 378
  airplane, 630, 638
  Bunnell, 709
  burn, 615–616
  cockup, 709–710
  finger, 615
  hand, 705–707
  ratchet, 709
  shin, 395–396
Split socket
  for below-elbow prostheses, 53
Split-thickness skin grafts (STSGs), 589, 691–693
Split Toe Flex Foot, 117–118
Spondylolisthesis, 385
Spondylolysis, 385–386
Spondylosis, 381
Spontaneous activity, 454–456, 494
Sport garments
  elastic, 665
Sports
  brachial plexopathies associated with, 497–499
  foot and ankle nerve injuries caused by, 531–532, 538, 540, 543–544
  peroneal neuropathies associated with, 529
  See also Exercise; *specific sport*
Sprains
  cervical, 361
  See also Ankle sprains
Spring balances, 802
Springlite Chopart prosthesis, 90
Springlite Company, 90
Springlite II, 119–120
Springlite Syme's prosthetic foot, 92
Springlite toe fillers, 89
Sprint runs
  maximal, 801
Spurling's maneuver, 364
SSRIs
  See Selective serotonin reuptake inhibitors (SSRIs)
Standing balance
  with knee-ankle-foot orthoses, 730
Standing table exercise, 603–604
Stapedial reflex, 312
  testing, 320
Stasis dermatitis
  after amputation, 137–138
Static orthosis
  for shoulder/elbow support, 713
Static strength
  measurement, 802
Static stretch, 359
Stationary attachment, flexible endoskeleton (SAFE) prosthetic foot, 92, 114–115
Stationary bicycles, 125, 635, 800–802
Steam burns, 577
STEN (STored ENergy) Foot, 120
Step length
  with ankle-foot orthoses, 720
Step-up hinges, 44, 53
Sternocleidomastoid muscle
  and accessory neuropathies, 336–338
Steroids
  for carpal tunnel syndrome, 513
  for cranial neuropathies, 280, 291, 315
  for digital neuropathies, 544
  for superficial peroneal neuropathy, 541–542
  See also specific drug
Stimulants
  for spinal cord injury, 167
  for traumatic brain injury, 214–215, 219
  See also specific drug
Stingers
  See Brachial plexus injuries
Stool softeners
  for immobilized patients, 753
Strain
  cervical, 361, 843–844
  in concussive brain injuries, 230
Strength, 782, 802–803
  correlation between muscle mass and, 795–796, 803
  definition, 802
  determinants, 803
  dynamic, 802
  gender differences, 806
  immobility-associated loss, 743–744
  measurement, 802–803, 806
  population data, 806
  static, 802
Strength detraining, 808
Strength-duration relationship
  in motor neurons, 449–450
Strength training, 813
  basic requirements, 468
  for burn patients, 635–636
  in integrated program, 813–814
  for musculoskeletal injuries, 359–360, 363, 367, 369–372, 375–377
  for peripheral nerve injuries, 467–470
Stress
  battle, 16, 623
  control principles, 623
  heat, 818–819
Stress fractures, 396–397, 816–818
Stress gastritis
  in spinal cord injured patients, 184–185
Stress ulcers, 753–755
Stretch
  elastic, 465

- plastic, 465
- prolonged, 603, 636, 746
- terminal, 604

Stretching exercises
- for burn patients, 628, 636, 652
- heating modalities used with, 467, 746–747
- for musculoskeletal injuries, 371, 373, 390–391, 398–399
- for peripheral neuropathies, 465–467

Stretch neuropathies, 440–443
- brachial plexus, 498–499
- clinical considerations, 440–441
- pathophysiology, 441–442
- peroneal nerve, 528
- prognosis, 442–443

Strickland, Benjamin A., Jr., 5, 9
Stroke volume (SV), 790–791, 801, 809
Strong-Campbell Interest Inventory, 195
Strong Interest Inventory (SII), 854
Structured Clinical Interview, 223
Stryker turning frame, 186
STSG
- *See* Split thickness skin graft (STSGs)

Stubbie prostheses, 133, 148–149
Stump edema syndrome, 134, 138
Stump wrapping
- complications, 125
- for lower extremity amputations, 87–88, 94, 96–98, 103–105, 125
- for upper extremity amputations, 48–50

Stuttering
- after traumatic brain injury, 222

Subclavius nerve, 496
Substance abuse
- and pharmacologic therapy, 240
- and traumatic brain injury, 229, 239–241, 246

Substance P inhibitors
- for peripheral nerve injuries, 478

Succinylcholine
- contraindications, 183

Sucralfate, 754
Suction sockets
- for above-elbow prostheses, 56
- for above-knee prostheses, 131
- silicone, 112

Suction transtibial prostheses, 111
Sudden infant death syndrome (SIDS), 323
Suicide
- by spinal cord injured patients, 196

Sulfamethoxazole, 752
Sulfamyalon
- *See* Mafenide acetate

Summation, 785
Sunburn, 580–581
Sunderland's classification
- of peripheral nerve injuries, 429–431

Sun exposure
- and burn patients, 633, 636

Superficial peroneal nerve, 540
Superficial peroneal neuropathy, 540–542
Superficial radial nerve compression, 505
Superior mesenteric artery syndrome, 755
Superior oblique muscle
- innervation, 292–293

Superior orbital fissure syndrome, 303
Supersensitivity
- denervation, 435–436, 456

Supine positioning
- anticontracture, 608

Supported employment model
- for brain injured patients, 247–250

Support groups
- for above-knee amputees, 134
- family, 255
- for hearing impaired patients, 325
- for multiple amputees, 149

Support Systems International, Inc., 186–187
Supracondylar cuff suspension
- of below-knee prostheses, 110

Supracondylar process syndrome, 506
Suprapatellar-supracondylar patellar tendon bearing socket, 109
Suprascapular nerve, 496
- injury to, 498–499

Sural entrapment neuropathy, 542–543
Sural nerve, 542
Surface electrodes, 453, 456–457
Surgical complications
- of lower extremity amputations, 134–148

Surgical procedures
- for above-knee amputations, 122–123
- for amputations, 45–46
- for anterior interosseous syndrome, 509
- for below-knee amputations, 93–96
- for brachial plexopathies, 497
- brachial plexopathies caused by, 499
- for burn wounds, 587–595, 628
- for carpal tunnel syndrome, 513–514
- for cranial neuropathies, 308, 335
- cranial neuropathies caused by, 280, 303, 332, 336
- delayed, 83, 465
- for digital neuropathies, 544–545
- for lower extremity amputations, 83–84
- neuromonitoring during, 280
- for peripheral neuropathies, 465
- peroneal neuropathies caused by, 529
- for phantom limb pain, 146–148
- for posterior interosseous nerve entrapment, 505
- for pressure sores, 187, 758
- safety factors, 628
- for spinal cord injury, 163–165
- for superficial peroneal neuropathy, 542
- for tarsal tunnel syndrome, 536
- for thoracic outlet syndrome, 502
- for ulnar neuropathy, 519
- for upper extremity amputations, 45–46
- *See also specific procedure or injury*

Surgical staples
- for skin grafting, 590

Surgical team
- multispecialty, 83

Suspension
- above-elbow prostheses, 57–58
- above-knee prostheses, 131
- below-elbow prostheses, 54–56
- below-knee prostheses, 110–112
- humeral neck amputation prostheses, 60
- immediate postoperative prostheses, 97–100
- suction, 112, 131
- supracondylar, 108

Suspension beds
- air, 757

Suspension belts
- for above-knee prostheses, 131
- for below-knee prostheses, 97–100, 110–111
- total elastic, 131
- transtibial prostheses, 131

Suspension hoops
  shoulder bypass, 714–715
Suspension sling arm support, 712–713
SV
  See Stroke volume (SV)
Swallowing, 326–328
  dysfunction, 327–328, 332, 334–335
Swallowing reflex, 327
Swan-Ganz line, 584–585
Swan neck deformity, 639
Swimming
  by burn patients, 636
Swing-through gait, 178
  with knee-ankle-foot orthoses, 729–731
SWOB
  See Separated without benefits (SWOB)
SWSP
  See Separated with severance pay (SWSP)
Syme's amputations, 82
  complications, 87
  functional outcome, 83
  joint contractures, 88
  long-term follow-up, 92
  prosthetic fitting and training, 87, 89–92
  rehabilitation, 87–89
  surgical technique, 87
  weight bearing, 91–92
Syme's sockets, 91–92
Sympathectomy
  for causalgia, 490
Sympathetically maintained pain syndromes, 428, 481
Sympathetic blockade
  for causalgia, 489–490
  for reflex sympathetic dystrophy, 141, 148
Sympathetic nervous system
  causalgia-associated changes in, 484–485
Symptom Checklist-90 (SCL-90), 253
Synaptic function
  modification, 212
Synaptogenesis
  reactive, 167, 212–213
Syndrome of Kiloh and Nevin
  See Anterior interosseous syndrome
Synkinesis, 315
Synthetic dressings
  for skin grafts, 593–594
Syringomyelia
  posttraumatic, 192
Syrinx
  acute posttraumatic, 173

T

Tachycardia, 333
Tacrine
  See Tetrahydro-9-aminoacridine (THA)
Tangential excision
  burn wound, 587–588
Tapia's syndrome, 340
Tardy ulnar palsy, 514, 516
Tarsal tunnel, 532–533
  anterior, 538
Tarsal tunnel syndrome, 531–536
  anatomic considerations, 532–533
  anterior, 538–540
  clinical presentation, 534–535
  differential diagnosis, 535
  electrodiagnosis, 535
  etiology, 533–534
  treatment, 535–536
Tarsometatarsal amputations, 85
  disadvantages, 83
  prosthetic fitting and training, 90
  surgical technique, 86
Tarsotarsal amputations, 85
  disadvantages, 83
  surgical technique, 86–87
Taste buds, 310–311
Taste sensation, 310–311
  loss, 312
Taylor, N. L., 742
Tay-Sachs disease, 287
TBI
  See Traumatic brain injury (TBI)
TBSA
  See Total body surface area (TBSA)
TCL
  See Tibial collateral ligament (TCL)
TC-3 socket, 131–132
TDRL
  See Temporary Disability Retired List (TDRL)
TDs
  See Terminal devices (TDs)
Tearing
  excessive, 312
Tears
  artificial, 315
Technetium 99m bone scintigraphy
  in causalgia, 487–488, 491
Tegaderm, 593–594, 670
Tegapore, 591, 594
Telephone adaptations, 620
Telescoping
  of phantom limb sensation, 88, 143
Temazepam, 220
Temperature
  high ambient, 818–819
  wet-bulb globe, 819
  See also Body temperature; Cold exposure; Heating modalities
Temporary Disability Retired List (TDRL), 865, 873–874, 876, 883
Tendinitis, 355
  Achilles, 399–400
  rehabilitation, 355
  rotator cuff, 369
  in upper extremity amputees, 61
Tendinosis, 355
Tendons
  function, 354
  healing phases, 354–355
  injuries, 354–355
  resistance training-induced changes in, 810
Tendon transfers, 179
Tenodesis grip, 179
Tenosynovitis
  De Quervain's, 376–377
Ten repetition maximum (TRM), 469
TENS
  See Transcutaneous electrical nerve stimulation (TENS)
Tensiometer
  cable, 802
Terminal devices (TDs)
  below-elbow prostheses, 44, 53–56
  body-powered, 53, 56
  hand, 53

  hook, 53
  myoelectric, 53, 56, 59
  recreational, 53–54
  selection of, 70–71
  vocational, 54
  World War II-era, 35
  See also Control systems
Terminal stretching, 604
TES belt
  See Total elastic suspension (TES) belt
Tetanus, 785
Tetrahydro-9-aminoacridine (THA), 216, 218–219
Teufel orthosis, 723–725
THA
  See Tetrahydro-9-aminoacridine (THA)
Theraband, 369–370, 374
Thermal injuries, 577
  combined with electrical burns, 578–579
Thermography
  in causalgia, 488
Thermoplastic materials
  in above-knee prostheses, 130–131, 133
  in below-knee prostheses, 109–110
  in finger prostheses, 39
Thigh corsets
  with side joints, 110–111
Thiothexene, 220
Thomas M. England General Hospital, New Jersey, 80
Thoracic outlet syndrome (TOS), 192, 366–367, 500–502
  classification, 500
  clinical presentation, 501
  differential diagnosis, 366
  electrodiagnosis, 501–502
  etiology, 500–501
  rehabilitation, 367
  treatment, 502
Thoracic spine injuries, 169
  complications, 182, 193
Thoracolumbar fractures, 174–175
Thoracolumbosacral orthosis (TLSO), 175
Thorax vascular support garment, 661
Three-jaw chuck grasp, 40–41
Three-state control systems
  for myoelectric prostheses, 66
Throat pain, 332–333
Thromboembolism, 762–764
Thrombolytic therapy, 762, 764
Through-knee amputations, 122–134
  prostheses, 132
Thumb
  amputation levels, 37
  preservation, 36–37
  reconstruction, 36–37, 39
  scar compression, 671–672
  ulnar collateral ligament injury, 377–378
Thumb prostheses, 39–41
Thumb spica cast, 377
Thyroarytenoid muscle, 333
Thyroid ophthalmopathy, 296
Thyrotropin-releasing hormone, 217
Tibial beveling
  inadequate, 140
Tibial collateral ligament (TCL), 393–394
Tibial fractures
  stress, 397
  type IIIC, 82–83
Tibial nerve, 532
Tibial neuropathy
  and ankle-foot orthoses use, 718–720
  case scenario, 493–496
  See also Tarsal tunnel syndrome
Tic douloureux, 280, 303, 308
  of glossopharyngeal nerve, 326, 328–329
Tics
  after traumatic brain injury, 220
Tilt-in-space recline wheelchair, 177
Tilt table, 175, 603–604, 765
Tincture of benzoin, 656–657
Tinel's sign, 140
  in anterior tarsal tunnel syndrome, 539
  in carpal tunnel syndrome, 511
  in superficial peroneal neuropathy, 541
  in tarsal tunnel syndrome, 534
  in ulnar neuropathy, 515
Tinnitus, 318, 323
Tissue complex injury, 356
Tissue overload complex, 356–357
Tissue proliferation
  on residual limb, 136–137
TKA line
  See Trochanter, knee, ankle (TKA) line
TLSO
  See Thoracolumbosacral orthosis (TLSO)
Toe amputations, 85
  prosthetic fitting and training, 89
  surgical technique, 85
Toe drag
  with plastic ankle-foot orthoses, 723
Toe fillers
  for shoes, 89
Toe flexion orthosis, 643
Toe transfer
  for thumb amputation, 37
Toileting
  adaptive aids, 679
  See also Activities of daily living (ADL)
Tokyo Metropolitan Rehabilitation Center socket, 131–132
Tolosa-Hunt syndrome, 295
Tongue
  disorders, 340
  evaluation, 340–341
  exercises, 341–342
  numbness, 304
  sensory innervation, 310
TOS
  See Thoracic outlet syndrome (TOS)
Total Army Personnel Command (PERSCOM), 866–867
Total body surface area (TBSA), 579–580
  estimation, 580
Total elastic suspension (TES) belt, 131
Total peripheral resistance (TPR), 790–791
Tourniquet injuries, 690
Tracheostomy
  after spinal cord injury, 181
  in burn patients, 599–600
Traction
  for back pain, 384–385
  halo skeletal, 630
  neuropathies associated with, 440–443, 498
Traction frame
  for upper extremity amputations, 45–46
Transcarpal amputations, 44
Transcutaneous electrical nerve stimulation (TENS)
  acupuncture-like, 480
  for burn pain management, 623
  complications, 480–481

  conventional, 480
  for musculoskeletal disorders, 358
  for peripheral nerve injuries, 479–481, 490
  for phantom limb pain, 142, 147–148
  for residual limb pain, 142
  for spinal cord injured patients, 191
  technique, 480
Transfemoral amputations, 122, 124
  bilateral, 122, 133, 148–149
  indications, 123
  surgical technique, 124
Transfemoral sockets, 124, 129–131
Transferable skills assessment
  in vocational rehabilitation, 855–856
Transfer training
  for amputees, 102–104, 106
  for spinal cord injured patients, 175, 189
Transmetacarpal amputations, 44
Transmetatarsal amputations
  disadvantages, 86
  functional outcome, 83
  prosthetic fitting and training, 89–90
  surgical technique, 86
Transpelvic amputations
  socket design, 132–133
Transphalangeal amputations, 44
Transportation
  skin traction systems for, 45–46, 83, 124
Transtibial amputations, 95
  complications, 101
  indications, 123
  rehabilitation, 102, 106
Transtibial prostheses
  suction, 111, 131
  suspension belts, 131
Transverse amputations
  prostheses, 39
Trapezius muscle
  and accessory neuropathies, 337–338
Traumacal, 602
Traumatic brain injury (TBI), 207–277
  and alcohol use, 229, 239–241, 246
  appetite dysregulation after, 217
  ataxia after, 217
  autonomic dysregulation after, 217
  behavioral dysfunction after, 212–213, 217–219, 222–223, 228, 245–246
  case management, 228–229
  classification, 208–209, 230
  closed or nonpenetrating, 208–209
  cognitive dysfunction after, 217–219, 222–223, 228, 237–238, 245–246
  community based rehabilitation, 228–229
  definition, 230
  diagnosis, 230–231, 234
  early recovery management programs, 226–227
  emergency medical treatment, 225
  epidemiology, 209–210
  etiology, 208–210
  family education, 227, 255
  family outcome, 227–228, 231, 245, 250–255
  functional outcome, 211–213, 224
  versus head injuries, 208, 231
  home based rehabilitation, 228
  incidence, 229
  interdisciplinary team management case study, 837–841
  low level neurologic states, 234–235
  management, 226, 228, 230–234
  mild, 229–234, 250
  military rehabilitation, 229–237
  model systems continuum of care, 225–229
  morbidity, 226, 234, 240
  movement disorders after, 220–221
  neural recovery mechanisms, 212–213
  neurobehavioral assessment, 231, 237, 241–245
  neurobehavioral outcome, 237–246
  neurobehavioral programs, 228
  neurogenic heterotopic ossification after, 221
  neuromedical issues, 217, 222–224
  nomenclature, 208–209
  ocular complications, 295, 299
  open or penetrating, 208–210
  orthoses, 704–740
  outpatient clinical services, 227–228
  outpatient rehabilitation team, 227
  Persian Gulf War, 15
  pharmacologic treatment, 214–222, 232–233
  prevention, 255
  prognostication issues, 210–212
  psychosocial outcome, 238–241
  rehabilitation, 226–227
  rehabilitation team, 225–229
  seizures after, 221–222
  sexual dysfunction after, 222
  social concerns, 235
  speech and language disorders after, 222
  surgical treatment, 225–226
  vocational rehabilitation, 228, 243, 246–250, 856
Trazodone hydrochloride, 215–216, 220–221, 476–477
Treadmills
  motor-driven, 800
Tremors
  after traumatic brain injury, 220
Trenchfoot, 443–446
Triamcinolone, 358
Triasmus, 304
Triceps pad, 55
Tricyclic antidepressants
  dosage, 476
  for peripheral neuropathy, 475–477, 491–492
  for phantom limb pain, 146, 148
  for reflex sympathetic dystrophy, 141–142
  side effects, 476
  for spinal cord injury, 191
  for traumatic brain injury, 214–215, 220, 222, 240
  See also specific drug
Trigeminal evoked potentials, 307–308
Trigeminal nerve, 300–308
  anatomy and function, 300–303
  reflexes involving, 302–303, 305–307
Trigeminal neuralgia
  See Tic douloureux
Trigeminal neuropathies, 303–304
  electrodiagnosis, 305
  evaluation, 304–305
  management, 308
  symptoms and signs, 304
  syndromes, 303–304
Trigeminal sensory neuropathy, 303
Triggering, 463
Trigger point injections, 382
Trimethoprim, 752
TRM
  See Ten repetition maximum (TRM)
Trochanter, knee, ankle (TKA) line, 127–128
Trochanteric bursitis, 389

Trochlear nerve, 291–300
  anatomy and function, 291–292
Trochlear neuropathies, 295–300
  electrodiagnosis, 299
  evaluation, 299
  imaging, 299–300
  management, 299–300
  symptoms and signs, 297–299
  syndromes, 296–297
Tropomyosin, 783
Troponin, 783
Truman, Harry S, 22
Trunk
  scar compression, 674
L-tryptophan, 216–217, 221
Tube feeding
  of burn patients, 602
Tubigrip external vascular supports, 657–660, 667
Tubiton Oedema sleeves, 613, 658–659
Tumors
  brainstem, 323
  cranial neuropathies secondary to, 280, 296, 317–318
Turkey, 28
Twitch, 785
Two-state control systems
  for myoelectric prostheses, 65–66
Tympanometry, 320

U

UCL-BC
  See University College of London Bioengineering Center (UCL-BC)
UDS
  See Utah Dynamic Socket (UDS)
UE amputation
  See Upper extremity (UE) amputations
Ulcerations
  of residual limb, 136
Ulcers
  in spinal cord injured patients, 184–185
  stress, 753–755
  See also Pressure sores
Ulnar amputations
  definition, 40
  prostheses, 42–43
Ulnar collateral ligament injury, 377–378
Ulnar nerve, 514, 519–520
Ulnar neuropathy, 438, 514–522
  anatomic considerations, 514–515, 519–520
  in burn patients, 689
  case scenario, 491–493
  clinical presentation, 515–516, 520–521
  differential diagnosis, 517, 521
  at elbow, 514–519
  electrodiagnosis, 514, 517–519, 521–522
  etiology, 516, 521
  treatment, 519, 522
  at wrist, 519–522
Ultrasound
  Doppler color-flow, 763
  in musculoskeletal injuries, 374, 377
  socket design with, 121, 130
Ultrasound diathermy, 467, 746–747, 758
Underwater weighing, 797–799
Unit assignment
  and aerobic fitness, 804
  and disability compensation evaluation, 869–872
  and vocational rehabilitation, 847
Unit dB, 320
United Kingdom
  military pension laws, 864
  Royal Air Force, 7, 16, 71, 150
  spinal cord injury units, 162
United Nations (UN), 25
Universal Below-the-Knee Bicycle Attachment, 125
University College of London Bioengineering Center (UCL-BC), 121
Unna dressings, 615, 632, 645, 656, 658–659
Upper extremity
  anticontracture positioning, 746
  scar compression, 671–673
Upper extremity (UE) amputations, 33–77
  activities of daily living, 50–52, 62–64
  bilateral, 61–65
  myoelectric prosthesis use, 68–69
  nerve recovery in, 82
  nomenclature and functional levels, 43–45
  psychological support, 47, 52
  rehabilitation, 46–52
  skin traction systems, 45
  surgery principles, 45–46
  vocational rehabilitation, 38, 41–42, 47, 52, 54, 71–73
  World War II, 34–35
  See also Above-elbow (AE) amputations; Below-elbow (BE) amputations; *specific type of amputation*
Upper extremity nerve injuries, 496–522
  See also specific injury
Upper extremity orthoses, 704–715
  See also specific type of orthosis
Upper extremity prostheses
  choice of, 69–71
  hybrid (myoelectric and body-powered), 56, 59
  post-Civil War era, 34
  postoperative fitting, 48–49
  and residual limb problems, 61
  training of amputee, 52, 65, 68–69
  See also specific type of prosthesis
Urinary catheters
  in burn patients, 585
  condom, 186–187
  in immobilized patients, 585
  in spinal cord injured patients, 193
Urinary incontinence
  in spinal cord injured patients, 193
Urinary retention
  in immobilized patients, 585, 752
  in spinal cord injured patients, 193–194
Urinary system
  effect of immobility on, 751–753
Urinary tract infections (UTIs)
  in immobilized patients, 751–752
  in spinal cord injured patients, 162, 181, 194–195
Ur-Nammu, 864
USAPDA
  See Army Physical Disability Agency (USAPDA)
USAR
  See Army Reserve (USAR)
Utah Dynamic Socket (UDS), 56
Utah myoelectric prosthesis, 67–68

V

Vagus nerve, 329–335
  anatomy and function, 329–331
  muscles innervated by, 331

  paralysis, 331
Vagus neuropathies, 331–335
  electrodiagnosis, 333–334
  evaluation, 333
  management, 334–335
  prognosis, 334
  symptoms and signs, 332–333
  syndromes, 332
VA hospital system
  See Veterans Affairs (VA) hospital system
Valgus, 716–717
  knee deformity, 736–737
Valium, 216, 220
Valleix phenomenon, 534
Valproic acid, 216, 220–221, 477
Valsalva's maneuver, 192–193
Vancouver General Hospital burn scar assessment, 662–664
VAPC shoe clasp orthosis, 722–723, 725
Vari-Flex Foot, 117–119
Varus, 716
  knee deformity, 736–737
Vascular access
  in burn patients, 584–585
Vascular innervation
  of peripheral nerves, 428
Vascular reconstruction, 82
Vascular studies
  for amputation complications, 135, 139
Vascular support
  after burn injury, 635, 645–649, 655–674
  for edema control, 471
  upper extremity, 671–673
Vascular support garments, 655–674
  adaptive aids for donning, 679–680
  custom-measured, 659–660, 665
  design, 661
  fitting, 661–662
  prefabricated, 665
  sources, 664–665
  use guidelines, 662
  See also specific type of garment
Vascular system
  of peripheral nerves, 427–428
  spinal cord injury-associated disruption, 166
Vasoconstriction, 791
Vasodilation, 791
Vasopressin, 218
VASRD
  See Veterans Administration Schedule for Rating Disabilities (VASRD)
Vegetative state
  definition, 234
  permanent, 234–235, 237
  persistent, 234–235
  prognosis, 211
  social concerns, 234–235
  stimulation programs, 235–237
Velcro closures
  for above-knee prostheses, 131–132
  for below-knee prostheses, 110
  for bilateral upper extremity amputees, 64
  for hand prostheses, 39–43
Velfoam, 667–668
Venography
  contrast, 762–763
Venous insufficiency
  after amputation, 137–138
Venous stasis
  and elastic wrap support, 601
Venous thrombosis
  See Deep venous thrombosis (DVT)
Ventilators, 181
Ventilatory equivalent, 789
Ventilatory regulation
  during exercise, 788–789, 801
Verapamil, 479
Vernet's syndrome, 326, 336
Verrucose hyperplasia
  of residual limb, 134, 138
Versed
  See Midazolam
Vertebral bodies, 167–168
Vertigo, 319, 324
  positional, 324
Vesicoureteral reflux
  in spinal cord injured patients, 193–194
Vestibular nerve
  anatomy and function, 317
  dysfunction, 319, 323–324
  evaluation, 321–322
Vestibular system, 294, 317
Vestibulocochlear nerve, 316
Vestibuloocular reflex, 292
  innervation, 295
Veterans Administration
  and physical disability system, 864–865, 873
  vocational rehabilitation program, 858–859
Veterans Administration Schedule for Rating Disabilities (VASRD), 868, 872–873
  rating codes, 874–875, 881
Veterans Affairs (VA) hospital system, 7–8, 34, 81
  brain injury treatment centers, 225–226
  spinal cord injury centers, 14, 196
Veterans Affairs Medical Center
  upper extremity amputee care, 50, 52
  vocational counselor, 47
Veterans Benefit Counselor, 859
Veterans Health Service
  kinesiotherapy, 831
Veterans Reserve Corps, 72
Vibration
  desensitization with, 633–634
Vicarious functioning
  after traumatic brain injury, 212
Vietnam Head Injury Study, 27, 210
Vietnam War
  amputations, 10, 52, 72, 80, 134, 148, 150
  causalgia, 482
  immobility complications, 742
  peripheral nerve injuries, 447, 497
  physical therapy, 23–24
  rehabilitation services, 7–10
  return to duty during, 9, 23–24, 150
  spinal cord injuries, 162
  vocational rehabilitation case study, 847–848
Vigabatrin, 222
Villaret's syndrome, 340
Vincamine, 219
Virchow-Robin spaces, 192
Visual acuity
  alteration, 287–289, 300
Visual evoked potentials, 290–291
Visual field defects, 287–289
Visual field testing, 288
Visual pathways, 287
  symptoms and signs, 288–289

- Visual perception, 294, 300
- Vitamins
  - for burn patients, 687
- VO$_2$
  - *See* Oxygen consumption (VO$_2$)
- Vocal cord paralysis, 332
- Vocational assessment, 850–857
  - for brain injured patients, 248–250
  - and educational experiences, 851
  - family, 853–854
  - functional, 852–853
  - interest, 854–855
  - interview, 853
  - measurement tool validity/reliability, 851–852
  - and medication effects, 851
  - and nature of disability, 851
  - and physical tolerance, 851
  - situational, 856
  - transferable skills, 855–856
- Vocational Interest, Experience, and Skill Assessment, 854
- Vocational outcomes
  - of amputees, 150
  - of brain injured patients, 234, 246–250
  - of spinal cord injured patients, 196
  - *See also* Return to duty
- Vocational rehabilitation, 845–861
  - for amputees, 150
  - for brain injured patients, 228, 243, 246–250, 856
  - case studies, 847–848, 850
  - coordination of necessary services for, 859
  - in-hospital phase, 846–857
  - for lower extremity amputees, 84, 93, 101, 106
  - posthospital phase, 858–859
  - resistance to, 850
  - short-term counseling in, 848–850
  - for spinal cord injured patients, 179–180, 195, 856
  - for upper extremity amputees, 38, 41–42, 47, 52, 54, 71–73
  - Veterans Affairs program, 858–859
  - Vietnam War case study, 847–848
  - *See also* Occupational therapy
- Vocational Rehabilitation Act Amendments, 6
- Vocational rehabilitation counselors
  - role in interdisciplinary team, 833
- Vogel, Emma, 21–23
- Voicaid, 620–621
- Voice impairments, 332
- Volar wrist flexion control orthosis, 709–710
- Volume of maximum oxygen consumption (VO$_2$max)
  - and cardiac output, 790
  - definition, 790
  - in immobilized patients, 765
  - measurement, 799–800
  - and muscle fatigue, 786
  - and oxygen transport variables, 792, 801
  - response to aerobic training, 809
  - values for elite athletes, 799
  - *See also* Aerobic capacity
- VO$_2$max
  - *See* Volume of maximum oxygen consumption (VO$_2$max)
- "V" straps
  - for above-elbow prostheses, 57–58

W

- Waist belt suspension
  - for above-knee prostheses, 131
  - for below-knee prostheses, 97–100, 110–111
- Walkers
  - for above-knee amputees, 127
  - for musculoskeletal injuries, 357
  - overhead, 604–605
- Walking
  - *See* Ambulation; Gait pattern
- Wallerian degeneration, 431–434
  - electrodiagnostic findings, 460
  - initiation, 432–433
- Walter Reed Army Medical Center, 7, 10, 21, 28, 34–35, 80
- Warm-up period
  - and muscle injury avoidance, 356
- War-related injuries
  - emotional reaction to, 846–848
- Water beds, 637
- Watershed infarctions, 211
- WDR neurons
  - *See* Wide dynamic range (WDR) neurons
- Weber's syndrome, 296
- Weber's test, 320
- Webril, 615, 630, 667
- Wechsler Adult Intelligence Scale, 195
- Wechsler Memory Scale-Local Memory subtest, 243
- Weighing
  - hydrostatic (underwater), 797–799
- Weight
  - of burn patient, 602, 688
  - and immobilization, 752
  - measurement, 793, 797–799
  - military control programs, 793
  - and muscle mass, 793
  - use for resistance training, 812
- Weight bearing
  - after above-knee amputations, 126
  - after below-knee amputations, 98–99, 102–103
  - after Syme's amputations, 91–92
  - and musculoskeletal injuries, 357
  - in spinal cord injured patients, 189
- Weight-bearing orthoses
  - ischial, 734–735
- Weight control programs
  - military services, 793, 795–797
  - physical training in, 793–794
- Weights
  - for training, 812
- Wernicke's syndrome, 296
- Western Neurosensory Stimulation Profile, 224
- Wet-bulb globe temperature (WBGT), 819
- Wheatstone bridge circuit
  - wire strain gauge with, 802
- Wheelchair, 682
  - controls, 177
  - manual, 177
  - power, 177
  - pressure sores caused by, 196
  - reclining, 175, 177
  - selection, 177
- Wheelchair cushions, 611
- Wheelchair locomotion
  - energy expenditure for, 731
- Wheelchair skills
  - patient education, 102–103, 175–177
- WHFO
  - *See* Wrist, hand, finger orthosis (WHFO)
- Whiplash, 171, 208, 230
- White matter, 165–166
  - damage, 165–166
- White phosphorus
  - burns caused by, 577–578

Widdowson, E. M., 742
Wide dynamic range (WDR) neurons, 484
Wide Range Achievement Test, 195
Wilbur, Ray Lyman, 5
Williams flexion exercises, 383–384, 391
Wire strain gauge
  with Wheatstone bridge circuit, 802
Women
  Korean War service, 23
  military body fat standards, 796, 798
  percent body fat values, 794
  physical capacity for exercise, 803–804
  reference body composition values, 792
  World War II service, 22
Women's Medical Specialist Corps (WMSC), 23
  See also Army Medical Specialist Corps (AMSC)
Word deafness, 320
Word discrimination test, 320
Work hardening, 858
Work hardening programs, 637
World War I
  amputations, 35, 81, 93, 95, 134
  cranial neuropathies, 332, 339
  disability retirement laws, 864
  musculoskeletal injuries, 11, 354
  peripheral nerve injuries, 420–421, 447
  physical reconstruction services, 4–5, 7–8
  physical therapy, 20–28, 830
  spinal cord injuries, 162
  trenchfoot, 443–446
World War II
  amputation centers, 7, 34–35, 52, 80–81, 149
  amputations, 34–35, 46, 71–72, 80, 93–95, 134
  causalgia, 481–482
  disability retirement laws, 864
  hand injuries, 35, 38
  immersion foot, 443–446
  immobility complications, 742
  musculoskeletal injuries, 11
  peripheral nerve injuries, 420–422, 447
  physical therapy, 21–22, 831
  rehabilitation services, 5–10
  spinal cord injuries, 162
  stress fractures, 817
Worth Four Dot flashlight, 299
Wound breakdown
  and immediate postoperative prostheses, 99, 102
Wound care
  burns, 596–607
  skin grafts, 590–591
Wound cleansing
  burns, 597–598, 627
Wound closure
  burns, 587
Wound contamination
  in lower extremity amputee, 81–83, 94–95, 134
  in upper extremity amputee, 46
Wound debridement
  See Debridement; Excision
Wound dehiscence
  after amputation, 135
Wound healing
  delayed, 102
  phases, 595–596
Wound maturation phase, 595–596, 631–684
Wound protection
  for lower extremity amputations, 87
  for upper extremity amputations, 48–50
Wright Linear Pump, 606–607, 665, 682
Wrist
  contracture reduction, 709, 746
  positioning for burn injuries, 610
  scar compression, 672
  strengthening exercises, 374
  ulnar neuropathy at, 519–522
Wrist, hand, finger orthosis (WHFO), 611–613, 630–631
Wrist disarticulation, 44
  prostheses, 44, 52
  and residual limb problems, 61
  surgical procedures, 46
Wrist disorders, 376–378
  See also Carpal tunnel syndrome (CTS); *specific disorder*
Wrist extension assist, 710
Wrist extensor-driven flexor hinge orthosis, 707–709
Wrist-flexion units, 54
Wrist-hand orthosis, 492, 746
  for carpal tunnel syndrome, 513
  for posterior interosseous nerve entrapment, 505
  for radial neuropathy, 504
Wrist orthoses, 709–710
  extension assist, 710
  volar flexion control, 709–710
Wrist units, 53–55, 57
  quick change, 54
  thin friction, 54
  variable-friction, 54
Writing
  adaptive devices, 621

X

Xeroform, 616, 638, 650

Y

Yohimbine, 222
Yom Kippur War, 15
"Y" strap
  for below-elbow prostheses, 55

Z

Zeiter, Walter J., 5
Zero-shear recline wheelchair, 177
Zimmer mesher, 590
Zinc oxide, 656
Zone of injury recovery, 167, 172–173
Zone of partial preservation, 169
Z-plasty, 691
"Z" strap attachment
  for above-elbow prostheses, 58–59